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PSYCHOTHERAPY IN MEDICAL PRACTICE*

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IT is a commonplace to say that psychotherapy is the oldest form of treatment. For, in pre-scientific days, when causes of disease were largely unknown, treatment must needs be empirical and in this treatment faith was a principal ingredient. In later times when one by one etiological factors were identified, the trend was naturally toward specific therapy directed to specific causes, and *pari passu* to discount and discredit psychotherapy or faith healing.

But as the concepts of health and disease broadened during the 19th century, and pathology which originally meant only tissue changes came to include psychic changes as well—that is psycho-pathology—it was inevitable that psychic factors should be ranged alongside physical factors in the causation of disability. That means that psychotherapy had to come to the fore again as a legitimate mode of treatment, but now on a different level corresponding to a better understanding of mental processes in health and illness. This new movement in what we may call scientific psychotherapy was initiated in France by Charcot, Bernheim and Janet.

It was a natural consequence that various systems or schools arose. The most conspicuous, shall we say notorious of these, was launched by Freud in Vienna. Without discounting Freud's contribution, it is necessary to say that his system soon took on all the characteristics of a religious cult with its passwords, its private, faintly mystical techniques, and an initiation ceremony vastly more comlicated and time-consuming, not to say expensive, than induction into the ancient mysteries or into present day fraternal orders—and with more dubious benefits.

But the psychotherapy we are concerned with in this discussion has naught to do with schools. Rather it is a simple procedure, universally applicable in medicine, and is essentially only applied common sense. We may define it as the influence of one mind on another to promote health. To avoid confusing psychotherapy with so-called faith healing, we should perhaps qualify the definition and say: the scientifically directed influence of one mind on another to promote health. But "scientifically directed" does not mean learning any elaborate or esoteric procedure or acquiring a new lingo or accepting as facts the arbitrary theories of any school. It means simply in accordance with elementary psychological principles and common human reactions.

Psychotherapy begins then with the first contact with the patient; and in general medical work the method of conducting this opening session of listening to complaints, observing the patient's demeanour, taking the history and making the initial examination is perhaps its most important feature.

In each case, the first major question the physician tries to answer is: what portion of the total disability presented by this patient is somatic and what portion is psychological? Estimates of the psychological components of illness, based on surveys of miscellaneous medical cases both in doctors' offices and on hospital wards, have ranged as high as 80%; and it is a conservative statement that at least 50% of the persons seeking medical help will present psychic features that have a definite bearing on the case and that must be taken into account in any complete and satisfactory treatment program.

A circumstance to which we pay perhaps too little attention is that when a person feels ill

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his natural tendency is to ascribe his malaise to some disordered bodily condition. He incorporates it, so to speak. He translates the subjective symptoms into physical terms; and in applying to the physician, his complaints may tend to be misleading to the degree that he has focussed his attention on this or that part of his anatomy. Even when he says: "It's my nerves," he is apt to mean the physical structures we call nerves.

Another preliminary consideration to be borne in mind is the enormously widened concept of health that social medicine has taught us. No longer is health merely a question of a machine that is sound by all the available criteria. That this is a primary condition goes without saying; but we have learned that in its all-inclusive sense health means also wholesome relationships in all the activities and interests that make up the life of the individual.

Chief of these are his social relations in general and his domestic situation in particular; then his occupational setting and his economic status. There are of course other areas in the health field but it is fair to single these out as of paramount importance. In industrial medicine, for example, it is well known that domestic trouble, personal worries and difficulties of all sorts promote invalidism and absenteeism and decrease efficiency.

It is also a commonplace that it may occur to the person asking for the doctor's help to tell him of his family trouble, his painful feud with his neighbour, his grievance against his employer, his fear of financial disaster. Somewhere in this broad area of social factors may lie the chief cause of the patient's ailment, and if we are concerned only with physical findings we may miss it entirely and our symptomatic treatment will probably do little good. We are hardly justified as physicians today in saying that these wider areas lie outside our field.

For the psychotherapy we wish to apply therefore, we need to know more than the patient's organs. We want to become acquainted with him as a *person* in his social and material environment. We want to understand him sympathetically and make him feel that we are interested in *him* as well as in his malady. We want to know what he thinks about, and particularly what he is worrying about. This is the very essence of psychotherapy, or at least the indispensable preparation for its successful application.

All this takes time; but I believe we err if we say that we cannot afford to give this time. Moreover it is likely to prove in the long run an economical spending of time, for the indications—all of them—will sooner become clear, and that should ensure earlier and better results of treatment.

Above all, when findings from the physical examination are negative, we are not justified in telling the patient that there is nothing wrong with him. If a person *believes* he is ill, he *is* ill, though the illness may be quite other than he conceives it to be. There is a saying that if a person is so ill as to think he is ill when he is not ill, then he is very ill indeed. It sounds like a paradox, but I think the meaning is clear. It is so easy for a patient to get the impression that the doctor thinks his trouble is only "imaginary", with all the unpleasant and unfavourable implications of such a conclusion. Here it is of very real importance for us to make a clear distinction between "imaginary illness" and *illness of the imagination;* and it may be useful to help the patient to some appreciation of this distinction too. If he feels, perhaps owing to his sensitiveness, that his complaints are regarded skeptically or unsympathetically, he will be on the defensive in reporting them; and it becomes the part of medical strategy to try to break down this defence, not by frontal attack but by an all-out effort to see his case as the patient sees it, to gain that kind of insight into his state of mind designated by the word "empathy". The goal is to change the patient's mind, or rather to get him to change it; and if this goal is to be reached it is not by contradicting or discrediting but by sympathetic understanding. It may even be less important to make a *diagnosis* than to know the *autognosis* as a starting point.

This is not the place to attempt a detailed outline of psychotherapeutic technique. Naturally it must be adapted in each case to the individual patient. Its success depends on the one hand on the good will, co-operation and faith of the patient, and on the other, on the knowledge, authority and skill of the physician. "C'est la foi qui guérit", said Charcot. And the central element in psychotherapy is suggestion, whatever technique one employs; and each physician will most advantageously de-

velop his own procedure rather than adopt that of another or of some school or cult.

Suggestion is a most potent weapon. It occasionally works where least intended, and sometimes unfortunately, as in the case of the girl who believed that her left side was becoming atrophied and developed a sense of weakness on that side, because she had heard the doctor remark casually in course of the physical examination that her left breast was slightly smaller than the right. Being physicians, we strive to relieve symptoms: we have also to be on guard against causing them. A contrary effect of unintended suggestion occurred in a male patient who mistook the securing of an electroencephalogram for a treatment measure and reported that he felt very much better afterward.

In recent years various kinds of shock therapy have been employed in all sorts of nervous and mental troubles; but it is now realized that even with these drastic procedures—which unfortunately have sometimes been looked upon as panaceas—psychotherapy is a very necessary if not indeed the most important part of the treatment for those patients who show good results.

As we remarked at the outset psychotherapy of a single common sense type is applicable throughout the field of medicine, and it is safe to say that all good doctors use it. There are many patients however for whom it will be the main indication, after the physical status has been fully assessed and the personal side of the patient's history has been explored. There are for example the patients with subjective symptoms such as weakness, fatigability, insomnia, or various kinds of discomfort which sufferers describe indiscriminately as pain; and they want treatment directed specifically to the symptoms complained of.

When such symptoms are not based on organic disease the method of attack has to be thoughtfully worked out in each case. The terrors of insomnia, for example, have been dispelled in some instances with a minimum of hypnotics by persuading the patient that it is not so much the fact of wakefulness that menaces health as the *dread* of lying awake in the dark lonely hours and the *fear* of the consequences. There are many complaints in which a similar shift of attention from the symptom itself to the patient's attitude toward it will tend to lessen the gravity of the original complaint.

Persons unduly worried about minor heart conditions or elevated blood pressure; patients with a simple adenoma of the thyroid whose nervous symptoms, being of other origin, are not relieved by thyroidectomy; young women who have had a period of amenorrhœa following shock or fright, or when entering upon a new and difficult occupation such as nursing; women who approach the menopause with the preconceived idea that it is a time of trouble; unfortunate individuals who, perhaps as a result of childhood indoctrination, suffer from dietary phobias; persons with a certain proneness to invalid reactions, as well as those with morbid fears of cancer, syphilis, tuberculosis; or those who, whatever the cause, become too dependent upon the doctor—all these have need for carefully considered psychotherapy.

We referred earlier to a distinction between faith healing and psychotherapy. We may now mention the crucial differentiation. In faith healing the sufferer plays a passive part. He brings only his capacity for blind belief, and awaits the miracle. Capacity for belief or confidence in the physician is essential in scientific psychotherapy as well; but here the patient takes an active part in the treatment program under the doctor's guidance. In favourable cases the consciousness that he has helped himself get over his troubles fortifies his self-confidence and stabilizes rehabilitation. Depending upon the rapidity with which the patient is able to learn his lessons and take over his own case, psychotherapy should be strictly self-limiting. Any procedure that goes on and on, perhaps with daily sessions, for months or years seems to me less a therapeutic measure than in itself a morbid and objectionable process.

As has been indicated, suggestion is the active element in all psychotherapy. In what has just been said we emphasize also the *educational* nature of this treatment; indeed in many cases psychotherapy and re-education are nearly equivalent terms. The objectives are first to give the patient a better understanding of his case, and then to show him how, with more or less help, he can do something about it. Such re-education may be focussed in one case on promoting logical thought processes; in another, on ways of controlling emotional expression; in a third, combating hypersensitiveness

and achieving "tough mindedness"; in still another, on the business of modifying habit patterns and routine behaviour; in another, on training to disregard inconsequential discomforts, neurotic paræsthesias and psychalgias; and in all cases, on resolving doubts and fears and supplying the reassurance necessary to fortify the patient's sense of security, to enable him to apply to himself with satisfaction those brave British words "carry on". There are many simple chores of this kind.

We learn by experience. Sickness is an experience from which not only the doctor but also the patient should learn. Insofar as psychological features are involved, and appropriate psychotherapy is applied, the patient will come to know himself better, be able to live more comfortably with his fellows, do his work better, and see new meaning in the concept of self-discipline and self-direction.

RÉSUMÉ

La psychothérapie est l'influence qu'un esprit exerce scientifiquement sur un autre esprit afin d'améliorer la santé. Cette définition bien comprise est complète; elle est basée sur le sens commun et elle n'a que faire des diverses écoles et des rites compliqués. Son arme principale est la suggestion, qui s'exercera sur la personne entière du malade. Il s'agit de comprendre celui-ci et de l'engager à raconter ses ennuis; il s'agit de l'écouter avec attention. Il faudra ensuite reprendre avec lui la route périlleuse et le persuader que d'autres interprétations que la sienne sont à envisager. Il faudra enfin que le malade prenne une part active à sa guérison, sous la direction de son médecin. La psychothérapie est une rééducation dont les buts principaux sont que le malade se comprenne mieux et qu'il puisse y arriver en grande partie par ses propres moyens. JEAN SAUCIER

T.N.T. SICKNESS

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IT is well known that T.N.T. (trinitrotoluene) causes illness in workers who are sufficiently exposed. The nature of the illness and its clinical manifestations is less common knowledge.

This paper is based on clinical records in some 2,500 personal consultations with patients suffering from various degrees of T.N.T. sickness.

This study was carried out in conjunction with efforts to prevent or control T.N.T. sickness in a shell-filling plant. The measures taken to prevent T.N.T. sickness were so successful that the number of consultations for this condition dropped in about a year from an initial figure of 240 per week to less than 2 cases per month. At this stage certain clinical studies involving laboratory investigation into the cause of cyanosis in T.N.T. sickness had to be abandoned because of the lack of clinical material.

A special clinical chart was designed. This record card provided for the name of the patient, sex, age, payroll number, place of work, etc. On it were columns for every known and suspected sign and symptom of T.N.T. sickness and columns for most of the common laboratory procedures which might be used in exploring the blood and urine changes. In addition there were some spare columns for symptoms or tests which might turn up later. The clinical record card was so constructed that about 20 consultations could be recorded on the same card allowing easy comparison of all symptoms and laboratory findings as they changed from date to date.

T.N.T. RASH

Some workers are sensitive to T.N.T. and cannot work in it without developing a rash. The skin reaction is not related to T.N.T. sickness and is not associated with generalized symptoms. The rash develops first on the areas exposed to direct contact with T.N.T., that is, usually the hands, arms, face, neck and ankles.

The rash is usually of the papular type sometimes resembling sago granules. It is extremely itchy. Sensitivity varies greatly. Some workers develop a severe rash soon after coming in contact with T.N.T. Others never develop a rash no matter how prolonged the contact. Some develop a few spots on the fingers which disappear in a few days, without even breaking contact. Some cases after an early mild rash on the hands seem to become immune and never have a recurrence. Others seem to be sensitized by the first rash so that a second contact results in a violent rash.

There is no specific treatment for the rash. The essential thing is removal from contact and thorough cleansing of the skin and change of clothing. Mild lotions such as calamine lotion in the early stages followed by mild skin cream in the later stages give the best results. It is well to remember that not all rashes developed while working in T.N.T. are caused by T.N.T. Some assistance in making a diagnosis may be obtained from a patch test, using an alcoholic solution of T.N.T. The test is read in 24 hours. The skin sensitive case will usually show a

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reddish rash under the patch at the end of 24 hours.

T.N.T. SICKNESS

The chief medical problem in T.N.T. workers is sickness. This may or may not be due to T.N.T. but it is always blamed on T.N.T. by the worker, and often also by his family physician who is not familiar with T.N.T. sickness. Differentiation from other common ailments is not easy and requires considerable experience.

Most of the literature mentions three ways by which T.N.T. becomes absorbed, namely: "through the skin, by inhalation, and by ingestion". It has usually been contended that the main avenue of absorption is through the skin. I have seen so many cases of illness develop in workers exposed to hot fumes that I am now convinced that inhalation is the more common route by which toxic quantities of T.N.T. are absorbed. Ingestion is not an important route except in workers who chew tobacco or gum while at work or those who eat without first washing their hands.

Observation of so many patients has led me to the conclusion that certain symptoms mentioned in the literature have clinical significance in T.N.T. sickness while others have not. For instance, sneezing, burning of the eyes and dryness of the nose and throat are not symptoms of T.N.T. sickness but only indications of local irritation such as might be produced by any dust.

Diarrhœa, constipation and abdominal distension were not found by me to be more frequent in T.N.T. workers than in any other workers and, therefore, should not be considered definite signs of T.N.T. sickness. Vomiting has long been considered a sign of T.N.T. sickness. In our series vomiting was found so infrequently that I am inclined to think it should not be included among the signs of T.N.T. sickness. On the other hand nausea was so common that I came to rely on it, in the absence of vomiting, as an important positive symptom.

As my experience increased I became able to diagnose T.N.T. sickness in its early stages and take the proper course of action. Eventually, I found that I could diagnose the onset of T.N.T. sickness even before the patient suspected it.

Eleven symptoms were found to have distinct clinical significance. I am listing them below in the order of their frequency. I am also giving in percentage the number of times these symptoms appeared in our whole series.

	%		%
Loss of appetite	47	Breathlessness	22
Undue fatigue	35	Dizziness	17
Cough	30	Abnormal drowsiness	16
Nausea	25	Abdominal cramps	14
Headache	25	Muscle pains	8
Tightness in chest	23		

I have noted occasionally tremor or tingling in the fingers or other parts of the body. I have also encountered occasionally urinary frequency with no demonstrable abnormality in the urine. The loss of appetite which is such a frequent symptom is particularly significant because in healthy workers T.N.T. increases the appetite.

Associated with the above symptoms of T.N.T. sickness are certain prominent signs. The most prominent is low blood pressure which was found in 80% of the cases. In our series blood pressure was not recorded as low unless it was more than 10% under the usually accepted level for both systolic and diastolic for the age of the patient concerned. Low hæmoglobin was encountered in 44% of the cases. The patient was not considered to have low hæmoglobin unless his hæmoglobin was under 85%. Our readings were done by the Sahli dilution method but with calibration adjusted so that 100% represented 15.6 grams as checked with the Evelyn photoelectric colorimeter.

Cyanosis was present in 20% of cases and jaundice in 17% of the cases. Cyanosis was noted on the lips, ears and face but was always verified by examining the tongue to rule out a duskiness due to racial colouring. Jaundice was recorded as present whenever it was detectable in the sclera. With practice one becomes able to detect even small amounts of jaundice. My records show that I have detected jaundice in the sclera when the van den Bergh was as low as 0.80 mgm. per 100 c.c.

In my experience cyanosis and jaundice were not commonly encountered together. In fact T.N.T. sickness seems to follow one of two distinct patterns, which I will call the hepatic type and the anæmic type since in the first the effect on the liver is a prominent feature and in the latter anæmia is very common. In milder cases neither pattern predominates. The symptoms, loss of appetite, nausea, undue fatigue and abnormal drowsiness are common

to both types. The severe cases tend distinctly to one pattern or the other.

The hepatic type may develop into toxic jaundice without any cyanosis appearing, without drop in hæmoglobin, and without blood pressure changes. In fact, these signs are conspicuous by their absence. In this type the blood picture usually remains normal, with possibly slight tendency to hæmoconcentration. In the hepatic type the chief complaints are: loss of appetite, nausea, fatigue, and weakness which are the usual symptoms of hepatitis. The jaundice may be visible in the sclera only or may become intense in the skin depending on the severity of the liver involvement. The Van den Bergh reaction is indirect and will, likewise, vary with the degree of liver damage.

The anæmic type of T.N.T. sickness is associated with more signs and symptoms. There is a tendency toward anæmia. There may be a rapid drop in hæmoglobin with a parallel drop in red blood cells. The anæmia is one of the iron deficiency type and may be fairly well controlled by the administration of iron. When the drop in hæmoglobin is rapid there will usually be a slight increase in the reticulocyte count. However, symptoms may be present without manifest drop in the hæmoglobin. The most common finding in this type of T.N.T. sickness is low blood pressure. Cyanosis is less common and occurs in the anæmic type. Aside from the symptoms of loss of appetite and nausea which are common to both types of T.N.T. sickness the symptoms in the anæmic type are what might be encountered in any case of disturbed circulation. There is the common symptom of breathlessness but no abnormality can be found in the heart. There is a cough without expectoration. There is commonly a tight feeling in the chest yet examination of the lungs reveals nothing. There are commonly headache, dizziness and abnormal drowsiness. There are sometimes abdominal cramps unassociated with either constipation or diarrhœa. Muscle pains are complained of occasionally and over all is the very common complaint of intense fatigue.

Several attempts were made to determine the cause of cyanosis. It was thought that methæmoglobin might be the substance causing the cyanosis. We made quite a number of investigations and came to the conclusion that although methæmoglobin was sometimes slightly elevated it could not be the cause of the cyanosis. Oxygen capacity of the blood was found to be normal in T.N.T. workers. Oxygen and carbon dioxide content of venous blood was found to be normal in T.N.T. workers. Carbon dioxide combining power and pH of plasma was also found to be normal.

Before our experiments on cyanosis were complete T.N.T. sickness in our plant became a rarity and cyanosis disappeared, so I am unable to produce conclusive evidence of its cause. On two occasions the cyanosis was made to disappear for a few minutes by violent exercise. A few minutes after exercise it reappeared. One patient was given pituitrin to see if this would cause the cyanosis to disappear. There was no effect on the cyanosis but at the same time the pituitrin failed to raise the blood pressure. We had no opportunity to explore this question further but I am inclined to think that cyanosis is an indication of circulatory insufficiency and is due to the slowness of peripheral circulation. This conception seems compatible with the findings of low blood pressure, breathlessness, tightness in the chest and fatigue.

DIFFERENTIAL DIAGNOSIS

The most common conditions which resemble T.N.T. sickness are obstructive jaundice, prostatitis, ordinary colds and chest conditions, heart disease, rheumatic fever, gastro-enteritis, diabetes mellitus, neurasthenia and malingering.

The hepatic type of T.N.T. sickness may develop into more or less severe toxic jaundice or even acute yellow atrophy of the liver. It is difficult to distinguish T.N.T. sickness of the hepatic type from obstructive jaundice, for both may be painless and intensely jaundiced. Both may have clay coloured stools, and nausea, weakness and vomiting. Both may have a nearly normal blood picture. Both may have an enlarged liver. The high indirect van den Bergh in T.N.T. toxic jaundice and comparative lack of skin itchiness may be factors in favour of T.N.T. sickness. However, every case of jaundice appearing in a T.N.T. worker should be presumed to be caused by T.N.T. until proved otherwise.

Subacute prostatitis may resemble mild T.N.T. sickness in every respect even to mild jaundice. Tenderness in the prostate gland on rectal examination is the distinguishing feature.

Ordinary colds and chest conditions may be ruled out by the absence of ordinary signs of congestion and swelling and the absence of nose and throat discharges and expectoration. T.N.T. sickness may simulate heart disease in some respects but no signs of organic heart disease can be found. Rheumatic fever may be distinguished by the sedimentation rate. T.N.T. sickness does not increase the sedimentation rate.

T.N.T. sickness should never be confused with tuberculosis of the lungs. T.N.T. never causes changes in the x-ray picture of the lungs. I have never known T.N.T. to cause asthma in a patient who did not already have it.

Gastro-enteritis is often confused with T.N.T. sickness. It differs from T.N.T. sickness in its rapid onset. The nausea of gastro-enteritis is usually accompanied by vomiting, while in T.N.T. sickness nausea is common but rarely associated with vomiting. Diarrhœa is commonly associated with gastro-enteritis but it is not a symptom of T.N.T. sickness.

Diabetes mellitus may be distinguished by the blood sugar test. Neurasthenia closely resembles mild T.N.T. sickness of the anæmic type. However, it may be distinguished by the persistence of the symptoms. A patient will recover from mild T.N.T. sickness completely in 3 or 4 weeks, at the most, if he is out of contact with T.N.T. during that time.

Malingering is a problem we had to deal with. Many people have a fear of T.N.T. and will complain of symptoms which others complain of. With experience one can usually spot these cases because they have no laboratory findings to substantiate their claims and in conversation they usually emphasize the fact that they want to get out of T.N.T.

I would like to emphasize the point that I do not depend upon laboratory procedures to make a diagnosis. A diagnosis of T.N.T. sickness can usually be made on clinical signs and symptoms before laboratory findings become positive. Furthermore, it must be remembered that it is impossible for a person to develop T.N.T. sickness unless he has a fairly heavy exposure.

TREATMENT

The first step in treatment of T.N.T. sickness is removal from contact with T.N.T. Mild cases may continue at some other type of work. Only the more severe cases need go to bed. All that is necessary is a saline laxative to promote elimination, a good dyspepsia mixture and complete abstinence from alcohol. Iron in the form of ferrous sulphate is given when necessary.

For bed patients our hospital procedure was as follows: (1) Bath room privileges, if not too ill. (2) Thorough bath and shampoo daily for four days to remove from the skin all T.N.T. possible. Acetone was used about the nails of the hands and feet. (3) Low fat, high carbohydrate diet with adequate protein and calories. Citrus fruit drinks sweetened by corn syrup, at least 3 glasses in 24 hours in addition to regular meals. (4) Magnesium sulphate and citric acid solution ½ oz. every morning for 4 days and other medication as ordered.

Laboratory work.—(*a*) Routine urinalysis, recording of urine colour, Webster test and blood pressure daily. (*b*) Every 3rd day—hæmoglobin, red cell count, hæmatocrit reading, smear, icterus index and van den Bergh. (*c*) Other procedures as ordered. Severe jaundice cases require further treatment of the liver by intravenous glucose, etc., in the usual manner.

A selection of representative case histories is given below.

CASE 1 (ANÆMIC TYPE)

C.M., male, aged 30. Regular T.N.T. worker. On June 15, 1942, he complained of undue fatigue, cough, tight feeling in chest, burning of eyes and abdominal distension. Around this date his blood pressure was 118/76; Hgb. 82%. He remained at work in contact with T.N.T. and on June 29 he was showing some cyanosis. His blood pressure had dropped to 104 systolic, 58 diastolic; Hgb. had dropped to 70%. He was taken out of contact with T.N.T. on June 29 and treated with a stomach mixture and Fe. Sulph. gr. 3, t.i.d. On July 8 he had recovered from all symptoms. His blood pressure was 122/68 and he returned to work in T.N.T. During the time of his illness he remained at work away from T.N.T.

CASE 2 (HEPATIC TYPE)

J.G., male, aged 55, Government inspector—exposed to T.N.T. On September 21, 1942, he complained of undue fatigue, headache, and cough. He was showing jaundice in the sclera, and slight yellowing of the skin. His urine showed a trace of T.N.T. Blood pressure was 120/80; Hgb. 75%; sedimentation rate 4 mm. in one hour; van den Bergh 1.2 mgm. per 100 c.c.; white blood cells 10,750; blood chloride 588 mgm. per 100 c.c. He was taken out of T.N.T. immediately and permanently. He was off work for approximately two weeks. On September 29 his symptoms were cough, tightness in the chest, dryness in the nose and throat, vomiting, undue fatigue and breathlessness. On this date jaundice had disappeared. His Webster test was negative; blood pressure 100/80; Hgb. 78%; sedimentation rate 11; blood chloride 616 mgm.; white blood cells 6,200; serum albumen 4.0%; serum globulin 2.81%. On October 7th serum albumen was 4.48%; serum globulin 2.50%; serum chloride 592; blood pressure 132/78; Hgb. 90%; van den Bergh 0.3 mgm. per 100 c.c. On October 22, blood pressure 140/80; van den Bergh 0.5 mgm.; Hgb. 90%; white blood cells 7,850; red blood cells 4,710,000; serum chloride 614 mgm.; serum globulin 2.34% serum albumen 4.49%.

On October 27 he was feeling much better. On December 1 he again had symptoms of loss of appetite, nausea, vomiting, dizziness, headache, undue fatigue, breathlessness, some swelling of the hands and feet. He also complained of a trembling feeling throughout the body, felt weak and said he would sweat very easily. On this date his Hgb. was 84%. He was not showing

any jaundice, reticulocyte count was 0.6%. He was treated with a stomach mixture and Fe. Sulph. gr. 3, t.i.d. and 1 gr. digitalis daily. On December 8, he had recovered from all symptoms.

CASE 3 (TOXIC JAUNDICE)

C.A., male, aged 26. This man was employed from September 3, to September 24, 1942, pulling formers out of 40 mm. shells with exposure to T.N.T. dust. From September 24 to November 4, he was drawing off molten T.N.T. from the melter into large steam jacketted kettles. At this operation he was exposed to heavy concentrations of T.N.T. fumes.

He first reported sick to me on November 4, 1942, complaining of loss of appetite, nausea but no vomiting, abdominal cramps without either diarrhœa or constipation, undue fatigue, a tight feeling in the chest and shortness of breath. There was no cyanosis and no jaundice detectable in the sclera. The blood pressure was 115/60 and his hæmoglobin 92%.

A diagnosis of T.N.T. sickness was made and he was immediately taken out of contact with T.N.T., but kept at work in another part of the plant. He was seen again by me on November 7, 1942, when he complained of the same symptoms and still did not show any cyanosis or jaundice. By November 13, the tight feeling in the chest had disappeared and did not again appear. There was not then, or had there been any cyanosis during his illness. On November 13, he still had a poor appetite, abdominal cramps and nausea. At this time he was also vomiting a little. He was now becoming a little jaundiced in the sclera. His van den Bergh reaction was 3.5 units.

In this case the diagnosis of T.N.T. sickness was made, on clinical evidence, one week before the appearance of jaundice and elevation of the van den Bergh. It seems likely that early removal from contact with T.N.T. was an important factor in the favourable outcome of this case.

His symptoms were much the same when seen again on November 17 when he was admitted to hospital. After a day or two in bed all symptoms disappeared except for poor appetite, nausea and weakness. He retained these three symptoms throughout the increase and decrease of his jaundice and until well into February, 1943. At no time did he complain of itchiness of the skin.

He remained under my care at our hospital until November 24, 1942, when he was transferred to the Toronto General Hospital where he remained until January 18, 1943. The patient remained under my supervision after leaving the Toronto General Hospital until he was seen last on May 25, 1943. His temperature remained normal throughout his illness except during an attack of chicken pox while in the Toronto General Hospital.

His liver and spleen were both just palpable on November 24 and remained palpable until about January 18, 1943. The enlargement of the spleen disappeared earlier than that of the liver. When examined on November 24, the left lobe of the liver as judged by percussion was definitely small. On January 18, 1943, the left lobe of the liver was normal in size.

There was evidence of hæmoconcentration in this case. On November 4, 1942, his hæmoglobin was 92%. It gradually rose until on November 21 the hæmoglobin was 115% with a red count of 5,700,000 and a hæmatocrit reading of 61. The blood concentration gradually decreased until on January 9, 1943, the hæmoglobin was again 92% with a red cell count of 4,600,000. The period of greatest hæmoconcentration was between November 18 and 24. During this period the hæmoglobin was done daily and all tests were 110% or more. The volume index, colour index, saturation index and sedimentation test remained normal throughout the illness. The white cell count was slightly elevated, in several tests reaching as high as 12,000. There was a very slight relative increase in neutrophils but no significant changes in the blood smear. The most significant laboratory findings were shown by the van den Bergh reaction, which rose gradually from 3.5 on November 13, 1942, to 18 on November 24. On November 25 it had jumped to 40. It remained at 40 until November 30 when it dropped to 35. It continued to drop gradually until by January 17, 1943, it was 1.2.

The serum phosphatase was elevated on two occasions in November. Prothrombin time was found consistently elevated to about 24 seconds throughout December. Capillary resistance and bleeding times were normal but on November 24, 1942, coagulation time was 5 minutes, 45 seconds. Blood cholesterol done on December 1, 1942, was 214 mgm. Blood sugar, non-protein nitrogen and serum protein tests when done were found to be essentially normal. The galactose tolerance test gave a total excretion of 3.58 gm. on November 28 and 3.19 on December 7, 1942, and thereafter it was normal. The cephalin-cholesterol flocculation test was the last to return to normal.

Urine examinations made throughout the illness were normal for specific gravity, albumen and sugar. Bile was present during November and early part of December. A few pus cells and granular casts appeared in late November. Urobilin was present in small quantities on November 26 and 28.

The Webster test showed traces of T.N.T. in the urine as late as November 22, 1942. Usually the Webster test becomes negative in four or five days after contact with T.N.T. is terminated. In this case the Webster test showed a trace of T.N.T. for eighteen days after his last contact. It would seem that this man had an impaired capacity to excrete T.N.T. The stools were normal except that sometimes they contained less than the normal amount of bile pigment.

When last seen on May 25, 1943, he was complaining of a little pain in the vicinity of the liver which was believed to be due to his apprehension in regard to his liver rather than actual disease. Nothing abnormal could be found on examination.

He was considered able to do light work on March 8, 1943. The patient's diet before the onset of his illness was quite well balanced and included plenty of meat vegetables and fruit. During September, October and November up to the onset of his illness he received daily a tablet containing 1,000 units of vitamin B, and 100 mgm. of vitamin C. He said he always took a shower bath at the end of every shift that he worked in T.N.T.

There was nothing very significant about the treatment of this patient. He was kept strictly in bed and under close observation. Early he had daily thorough baths and shampoo to remove all possible T.N.T. He was given magnesium sulphate to ensure watery stools for the first few days. He was given a high carbohydrate, low fat diet with plenty of fluids and fruit juices. He was able to take adequate carbohydrates by mouth so that it was not necessary to give him glucose intravenously. He was given a moderate amount of protein and had brewer's yeast daily.

SUMMARY

We have outlined the signs and symptoms of T.N.T. sickness as encountered in some 2,500 consultations for this condition.

Some points are presented in differential diagnoses which we have found of value from personal experience.

A number of typical case histories and one severe case with recovery, are presented.

THE CONDUCT OF TRIAL OF LABOUR*

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THE time-honoured classification of cases of cephalopelvic disproportion into three main groups is an obvious one and in modern clinical practice it retains its usefulness. At one extreme are those patients in whom the disproportion is of a minor degree, when a safe delivery *per vias naturales* can be expected with confidence. In contrast are those where the amount of overlapping of the head at the brim is so great, or where the pelvic deformity is so marked that Cæsarean section is the only mode of delivery to be considered. As is usually the case, it is the intermediate group in which the decision is more difficult. One would expect that with increasing experience on the part of the obstetrician the number of cases falling in this group would diminish, and to a point this is true. Nevertheless I am convinced that there will always be an appreciable percentage of patients in whom the outcome of labour cannot be accurately foretold and to whom a trial of labour must be given. Even so eminent an obstetrician as Munro Kerr admits to a 6 to 8% error in his judgment as to whether a head will pass through a pelvis, and this, mark you, when the patient is examined early in labour, not at the thirty-eighth week, when so many obstetricians make their final decision.

It should be counted no reproach that in a case of moderate disproportion a definite opinion cannot be given on the prospect for vaginal delivery. On the other hand one distrusts the doctor whose confidence in his own judgment in this matter is complete. If such a man's practice is closely examined it will be found that he does a large number of elective Cæsarean sections for so-called disproportion. I am quite sure that a significant percentage of these would have proved unnecessary, had a trial labour been allowed.

It might be thought that the wide employment of radiology in obstetrics would eliminate the group of doubtful cases. Certainly, by giving us accurate measurements of the pelvic diameters and an exact knowledge of pelvic architecture in any given case, it is possible, before labour starts, to assign more cases to the groups of minor or major disproportion. But radiology does not provide the complete answer to the question because delivery is not an entirely mechanical problem. Radiology tells us only of structure and nothing of function. It has nothing to say of those two very important factors in the mechanism of labour, the force of the uterine contractions and the ability of the fetal head to mould past an obstruction. Therefore the knowledge supplied by a radiological study of the pelvis must be supplemented by the experience of the obstetrician, accustomed as he is to observe closely uterine action during labour. He is but a poor obstetrician who makes a decision as to the mode of delivery by merely glancing at a set of pelvic measurements furnished by the radiologist, and the latter would not claim to be the one who should decide.

A trial of labour, according to strict definition, is one which has progressed to full dilatation of the cervix and continued in the second stage for two hours. In actual practice these conditions are satisfied in only a few instances, since it is generally possible to tell at an earlier point whether the fetal head is going to pass successfully through the pelvis. After eight or twelve hours of strong contractions one is usually able to distinguish those cases which will require delivery by Cæsarean section. If, on the other hand, after such an interval or sooner, the fetal head has become engaged, the trial is at an end and the case is to be managed on ordinary lines. It must be emphasized that every trial of labour must be so conducted that operative intervention can be undertaken at any time with comparative safety to the mother. To interfere after labour has been so long in progress or so carelessly managed that gross infection of the genital tract is evident, means that one has delayed too long. In all cases it goes without saying that the lower segment section is the only one to be employed, or else an extra-peritoneal operation. Classical section can be performed with safety only after labour has been in progress too short a time to enable a true test of labour to be given.

At the outset it must be admitted that not all cases are suitable for a trial of labour. It is precluded, obviously, when the fetus presents

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by the breech. It will not be selected when other obstetrical abnormalities are associated with the disproportion, for example, placenta prævia, a severe grade of toxæmia or eclampsia, or an oblique lie which is not easily corrected and maintained as a vertex presentation. When the patient is an elderly primigravida or has had an unfortunate obstetric history there may well be some reluctance to elect a method which does incur a somewhat higher risk to the fetus. The fact that a patient has had a previous Cæsarean section does not necessarily preclude a trial of labour subsequently, though the danger of rupture of the old scar, especially if the operation was of the classical type, makes very close supervision imperative and the trial in such cases should not be prolonged. Should the patient suffer from asthma, cardiac disease or any other condition which makes her unfit for fairly strenuous muscular exertion, trial of labour in the presence of moderate disproportion is best avoided in favour of elective section.

Before the onset of labour a careful estimation of the amount of disproportion must be made. This examination should take place in the ante-natal clinic, preferably about the 38th week, and if labour does not commence at term the case should be reviewed at weekly intervals after the calculated date of delivery. The size of the pelvis should be determined by clinical methods. The usual external measurements can be taken provided it is realized that their significance is practically negligible. Of supreme importance is the digital examination of the pelvis with the patient in the lithotomy position, when the points to be noted are: the ease with which the sacral promontory can be felt, the amount of the pelvic brim accessible to palpation, the amount of room in the forepelvis, the size of the sub-pubic angle and of the intertuberischial diameter. On the same occasion an attempt is made to push the head down into the pelvic brim, when the amount of overlap is estimated by Munro Kerr's method of resting the thumb of the examining hand on the pubic symphysis. If the patient is too resistant to permit this examination, the use of an anæsthetic is justifiable. A simpler, but less effective method of gauging overlap is to palpate at the symphysis after the patient has risen from the supine to a semi-sitting position. In many cases this will suffice to push the fetal head well into the brim, as will also having her lie on her left side with both knees drawn up. Rectal examination may then demonstrate the fetal head in the pelvis.

Asynclitism of the fetal head may lead one astray in assessing the amount of disproportion by clinical methods. If there is anterior asynclitism, *i.e.*, if the presentation tends to be a posterior parietal one, the amount of overlap appears much more than it will be if and when the asynclitism is corrected. The reverse is also true; posterior asynclitism may give the impression that the head will push easily through the brim when actually serious disproportion is present. This source of error must be kept in mind.

If the results of clinical investigation are equivocal, the exact measurements of the pelvis should be determined by radiological methods, using any one of the many standard techniques. The radiologist will also be able to classify the pelvis according to the accepted types described by Caldwell and Molloy.

If the case is considered a suitable one for a trial of labour the patient should be permitted to go into labour spontaneously. One is often tempted to give a medical induction to these patients, especially if they go beyond term, in order to limit, at least to some degree, the size of the fetus. Frequently, however, the only effect of the induction, even when repeated small doses of pituitary extract are administered, is to cause premature rupture of the membranes. If, as so often happens, twenty-four hours or an even longer interval elapse before the onset of regular contractions, the risk to the mother, if a Cæsarean section should ultimately become necessary, is definitely increased.

The nursing care of the patient throughout the first stage follows the accepted principles governing the management of any woman in labour. Scrupulous attention to every detail is essential and complete notes should be kept. As well as a four-hourly temperature chart, the maternal pulse and the fetal heart rate should be taken at hourly intervals, and as labour advances the latter observation must be made even more often. Every effort should be made to keep the membranes intact as long as possible in order to decrease the risk of infection of the amniotic cavity and to this end it is better that the patient should remain in bed. The lower bowel must be kept empty and the bladder must be evacuated at regular intervals,

if necessary by catheter. The patient should be encouraged to take plenty of fluid by mouth and if dehydration becomes evident glucose solution should be administered intravenously. Sedatives are of the greatest importance but should not be given until contractions are well established. In spite of the present vogue for demerol I believe morphine gr. ¼ is still the most valuable drug we possess for cases of prolonged labour; it is often advisable to combine with it one of the short-acting barbiturates, such as seconal. The nursing staff, as well as the doctor in attendance, should realize the importance of maintaining the patient's general morale. If they understand the situation and why it is being thus managed, most women will co-operate well. Too much stress cannot be laid on the preservation of asepsis throughout labour, so necessary because of the possibility of surgical intervention. The risk attached to rectal examinations should be understood, so that these are not made indiscriminately but only when indicated, while vaginal examinations should be made only when the patient has been cleansed and draped and the doctor prepared as for delivery.

Once labour has definitely commenced, an estimation of progress must be made at repeated intervals and developments, favourable or otherwise, sought. The strength, duration and frequency of the contractions are taken into account, with the general condition of the mother and the fetus. Special attention, naturally, is paid to the level of the presenting part, whether or not it remains above the brim, and, if it attempts to engage, how much of the head has passed the plane of the inlet.

Abdominal examination yields much valuable information and of course can be made as often as necessary with impunity. By it one can follow the gradual descent of the cephalic prominences in favourable cases, and it enables one to determine the degree of flexion of the fetal head. When a head that was formerly mobile becomes fixed, one is by no means justified in assuming that the prospect of vaginal delivery is assured by that sign alone. Even with insuperable disproportion, strong contractions can so jam the head into the brim that it cannot be moved in performing Pawlik's grip. On the other hand, fixation may be the first indication that the head will successfully engage.

If one is uncertain, from abdominal examination, as to the level of the presenting part, this can be checked by rectal examination. It must be realized that one may be led astray in this particular by rectal examination in those cases where a large caput forms, since the level of the caput may be mistaken for that of the fetal head proper, and the difference between the two can be considerable. The information from a rectal examination, even when performed by an experienced obstetrician, is limited and is often incorrect. Therefore a vaginal examination is essential in most cases of trial of labour, the only exceptions being those where the head engages well after a short interval or else makes no attempt whatever to enter the brim. Provided it is done with full aseptic precautions, vaginal examination, even if repeated once, does not seriously increase the risk of infection to the patient if Cæsarean section ultimately proves necessary. As well as noting the amount of cervical dilatation and the station of the fetal head, one should determine also the position of the head in the pelvis, the amount of de-flexion present, the size of the caput succedaneum, the amount of moulding and whether the cervix is well applied to the presenting part. Vaginal examination is indicated when the membranes rupture, to exclude the presence of prolapse of the cord and later, to discover the cause of any delay in progress.

The finding of a so-called occipito-posterior position should not influence the management of a trial of labour. Too often labour has been terminated by a prematurely performed Cæsarean section because this diagnosis has been made. It should be recognized that in the great majority of cases the fetal head engages or attempts to engage with the sagittal suture in approximately the transverse diameter of the brim and descends in this so-called transverse position, not rotating anteriorly or posteriorly, as the case may be, until it is in the pelvic cavity late in the first stage or else failing to do so at all. When the head is found in the transverse position it is often mistaken for an occipito-posterior and an unfavourable prognosis is given, instead of the mechanism being recognized as a perfectly normal one.

How long should a trial of labour last? It should be realized at the outset that the development of signs of distress on the part of the mother or the fetus before engagement of

the fetal head, calls for immediate termination of the labour by Cæsarean section. This includes all cases in which signs of obstructed labour become evident. In the absence of such unfavourable circumstances it is safe to allow the patient to continue in active labour for twenty-four hours. By the end of this time, and often before, it is usually possible to make a decision as to the mode of delivery. If the head is not engaging well after this interval, it is unlikely ever to do so. Generally speaking, however, the application of rigid time limits to all cases is unwise. It is not a question of how many hours the patient has been in labour, but the amount of progress she is making in view of the strength and frequency of the contractions. It must be remembered, too, that it is a trial of what the mother can accomplish, not of what she can endure, and so the labour must not be permitted to drag on indefinitely.

No patient can be said to have had a thorough trial until the membranes have ruptured. Prior to this event the buoyancy of the liquor amnii may have prevented the fetal head from fitting down into the pelvic brim. If the patient reaches complete cervical dilatation it is justifiable to rupture the membranes artificially; thereafter the head may descend rapidly. If the head fails to advance after two or three hours of strong contractions with the membranes ruptured and the cervix partly dilated, particularly in the presence of a large caput, excessive moulding, or a cervix poorly applied to the presenting part, termination of the labour by Cæsarean section is called for.

In arriving at a final decision the type of pelvic contraction must be taken into account. If the pelvis is of the typical flat or platypelloid type, once the fetal head has passed the brim no further difficulty need be anticipated, since there is usually plenty of room in the cavity and at the outlet. On the other hand, if one has to deal with a generally contracted or small gynæcoid pelvis difficulty will be encountered at every level and the trial cannot be considered at an end until the fetal head is well down in the pelvic cavity. Where the contraction is chiefly at the outlet a trial of labour, strictly speaking, cannot be given; when it is found that the head cannot be brought through the outlet it is too late to adopt abdominal section. The degree of contraction must be carefully assessed and the mode of delivery decided on before the head reaches the lower straits. Incidentally, it is surprising through what a narrow intertuberischial diameter an average-sized fetal head will pass.

Once the head is in the pelvic cavity, forceps may be applied with safety, as in any labour, provided the usual conditions are fulfilled. The application of high forceps has no place in the conduct of a trial of labour; there is no question of assisting the passage of the head through the brim past an obstruction by the use of traction.

Not infrequently circumstances arise early in labour which render difficult the conduct of a thorough trial. If primary uterine inertia develops and persists for twenty-four hours, it is generally evident by that time that the poorly sustained and infrequent contractions will not suffice to overcome the degree of disproportion that is present. To allow labour to continue longer in the expectation that stronger pains will ensue involves some risk to the mother, should one's hopes be disappointed and a Cæsarean section be necessary after the patient has been three or four days in labour. The same considerations arise in those cases where the membranes rupture prematurely and contractions fail to develop. If twenty-four to thirty-six hours pass and labour is not yet established, one is forced to perform section when the outcome for vaginal delivery is still uncertain. One is naturally loath to try to induce pains by the use of pituitary extract when the head is above the brim, though the procedure is not especially dangerous when contractions have not yet started. The dose should never exceed 2 units, which should be repeated not oftener than at half-hourly intervals. The administration of synthetic œstrogens by mouth may be of value in some of these cases and is certainly worth trying. A suggested dosage is 5 to 10 mgm. stilbœstrol every hour until as much as 100 mgm. has been given. The occurrence of prolapse of the cord early in labour in patients selected for a trial obviously calls for the abandonment of the original plan and the immediate performance of section.

What of the management of subsequent labours in patients to whom a trial of labour has been given? If a vaginal delivery occurred with no undue difficulty, of a normal-sized child, then obviously on a future occasion the

same favourable outcome may be anticipated, while bearing in mind the possibility of a larger fetus in later pregnancies. On the other hand, if the patient ultimately had to be delivered by section, this mode of delivery should be selected again. In those patients who were delivered vaginally at a first confinement but because of disproportion only after a long and difficult labour, it is good obstetric practice to induce labour prematurely at 36 to 38 weeks when these mothers present themselves for management a second time. This, it must be pointed out, is not the same thing as treatment of disproportion by premature induction as an alternative to trial of labour.

In any series of cases subjected to a carefully managed and thorough trial of labour it will be found that a successful vaginal delivery can be conducted in the great majority. Indeed, it is surprising how few Cæsarean sections will be required. This applies only, however, if adequate time is given to see what the mother can accomplish. Too often a patient is permitted merely to start in labour, time is not allowed for effective contractions to develop, and the decision to perform a section is made before the membranes have ruptured. The management of a true trial of labour demands much from the obstetrician; he must see his patient at frequent intervals; he has to exercise much patience and restraint and in making the final decision he must summon all his experience and take into account every item of evidence gained from painstaking examination and observation of the case. It is obviously much easier for him to perform elective Cæsarean sections on all cases where there is a possibility of difficulty, but he will miss the satisfaction of knowing that he has spared the greater number of such mothers a major surgical operation which carries still a higher risk than vaginal delivery.

The total problem of chronic disease is not a series of separate problems which can be solved one by one, but rather a complex of interrelated problems which require simultaneous solution. Co-ordinated and comprehensive planning should be undertaken at all levels in order to achieve effective action to meet the challenge of chronic illness.—*Am. J. Pub. Health*, **37**: 1265, 1947.

THE DIAGNOSTIC PROBLEM IN POLIOMYELITIS*

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WHEN the Poliomyelitis Diagnostic Clinic of the Royal Victoria Hospital opened on August 1, 1946, there were no previous records to guide the interns in their work of examining suspects and recording their findings in an orderly way. The present summary of the experience of this clinic is presented in order that succeeding groups of interns, if called upon in another epidemic, may have the benefit of our hastily acquired experience and may profit from the mistakes which we made. In retrospect one realizes what important facts could have been ascertained, even from such a small clinic as we operated, if the present status of the diagnostic problem in poliomyelitis had been adequately understood by us from the beginning.

The clinic was set up to diagnose cases in the age group not covered by the Children's Memorial Hospital nearby, and to screen out the cases which were not poliomyelitis. The clinic was not limited entirely to adults, however, and several children were seen. No charge was made for any diagnostic procedure. Because of the hysteria generated in such an epidemic it was thought wise to question every case before admission to the actual examining room. There was no adequate method of rendering sterile this room and its instruments, and to avoid possible infection of the normals an interview in the admitting office seemed justified. In all, 133 suspects were examined in the clinic proper. Lumbar puncture was carried out routinely but was refused or made impossible in 4 cases.

RESULTS

Due to the changing intern personnel, the pressure of work at times, and the advanced state of the bulbar cases, the data collected were not always uniform. (In order that they may be, in future, a suggested questionnaire is

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appended to this paper.) All results are calculated as percentage of cases in which the particular datum was available. The four cases on whom lumbar puncture could not be done have been eliminated from the series, leaving a total of 129 cases. Of these, 57 cases were diagnosed as poliomyelitis when seen, and the remaining 72 as "not poliomyelitis". In retrospect, two of the latter cases might be placed in the poliomyelitis group. This matter will be taken up in the Discussion. It should be stated that cerebrospinal fluid pressures were worthless in this series as diagnostic aids, due to the lack of co-operation by some patients, both children and adults. The detailed analysis follows:

	57 cases diagnosed polio-myelitis	*72 cases diagnosed not polio-myelitis*
Males	56.0%	50.0%
Females	44.0%	50.0%
Average age	15.2 yrs.	18.7 yrs
Subjective:		
Sore throat	10.5%	20.8%
Anorexia	31.6%	26.0%
Nausea or vomiting	49.0%	41.6%
Headache	60.0%	50.0%
Muscle pain	61.4%	47.0%
Neck or back stiff or sore	90.0%	66.6%
Objective		
Av. temperature	100.7°F	100.1°F
Positive Kernig	35.0%	1.3%
Demonstrable muscle weakness	56.0%	6.9%
Unequal or decreased deep reflexes.	72.0%	8.0%
Av. C.S.F. cell count	161 cells	1.4 cells
Av. C.S.F. protein	61.7 mgm. %	33.0 mgm. %

The youngest case diagnosed poliomyelitis in this group was 2 years of age and the oldest 40 years.

Diarrhœa and somnolence were rarely encountered in this clinic. Of the 57 cases of poliomyelitis, 9 were of the bulbar type, 2 of these being serious and the remaining seven mild. Twenty-five of the 57 cases were non-paralytic in nature. No significant difference existed between these and paralytic cases in the matter of cerebrospinal fluid proteins or cell counts.

DIFFERENTIAL DIAGNOSIS

Some of the physicians who consulted the clinic wished to know what set of findings would establish the diagnosis as poliomyelitis, with less concern, possibly, for the diagnosis in the "left-overs". In this clinic more than half the suspects were diagnosed as "not poliomyelitis". The bulk of these cases were suffering from acute pharyngitis, grippe, or gastro-intestinal upsets. There was one case of subarachnoid hæmorrhage in a hypertensive man aged 53, who was regrettably brought 60 miles by ambulance for lack of a lumbar puncture at home. Two cases of ruptured intervertebral disc were diagnosed, while one case of post-inoculation urticarial reaction with muscle pain was seen. A case of sun-stroke, an acute torticollis and a post-febrile strabismus were to be found in the non-poliomyelitis cases.

The following cases are presented briefly, to emphasize some of the pitfalls which await one at the time of a poliomyelitis epidemic.

CASE 1

L.T., male, aged 34. This patient was admitted from Isle Perrot, 20 miles distant, with a history, given by his friends, of having been struck by a large auto-wrecking crane in the chest and scapular region one week previously. He was brought in because he was becoming "paralyzed" and had difficulty in urinating. On examination the man presented nothing like the usual picture for a high thoracic or cervical cord injury. Instead, he showed a very advanced bulbar palsy with cyanosis. He could walk about perfectly well and actively, but could not move his arms nor hold his head up. A lumbar puncture showed over 200 cells in his cerebrospinal fluid., Pandy positive, and cerebrospinal fluid proteins 73 mgm. %. Cyanosis increased rapidly and artificial respiration was maintained throughout a desperate ambulance run to the Alexandra Hospital. Despite the use of a respirator, the patient died an anoxic death within 6 hours. As a result of this episode, a foot-operated suction pump was borrowed from the Montreal Military Hospital and kept in the Alexandra Hospital's ambulance until a suction line was installed by their resourceful driver, to be operated from the windshield wiper's vacuum system. A large oxygen tank and mask were also placed in this ambulance for such emergencies.

CASE 6

B.G., female, aged 25. This patient was five months' pregnant at the onset of the disease. She showed unusual skin hyper-irritability, a positive Kernig sign, and excessive tenderness of limb muscles. Her C.S.F. contained 305 cells, 75% being polymorphonuclears. Despite these findings she did not go on to any muscle paralysis, and eventually had a normal, full-term child, which has a normal nervous system. Two features of this case will bear discussion. The painful skin sensitivity to touch was not marked in any other case, but was a common finding in the California epidemic of 1932.[1] The lack of transference of the disease to the fetus is in keeping with the reported experience of others. Weaver[2] reviewed 70 cases in the literature. In no instance was a paralyzed child born. He has shown in cotton rats that the Armstrong-Lansing strain of poliomyelitis virus is not transmitted by the mother to the fetus *in utero*, nor to the offspring by the mammary gland. McKhann and Chu[3] were able to prepare a placental extract which would neutralize poliomyelitis virus *in vitro*.

CASE 22

Mrs. M.F., aged 19. This patient was swimming in the St. Lawrence on July 21 and August 4 and developed respiratory difficulty and increasing pain and weakness of the limbs on August 12. It was subsequently discovered that at the end of July an Indian child had developed poliomyelitis in the village immediately above the beach on which Mrs. M.F. bathed. The sewage of the Indian hospital in which the child was quartered

drained directly into the St. Lawrence. This case may throw some light on the water-borne nature of the disease and the presumed incubation period of 8 to 14 days. Because of the patient's proximity to the Indian village tuberculous meningitis had to be ruled out. No bacteria could be stained, no pellicle formed on the C.S.F., and the chest x-ray was negative.

CASE 36

C.W., male, aged 15. On August 7 this boy was swimming in the St. Lawrence near Dorval. On August 15, while on a long canoe trip north of St. Jovite, he became tired and nauseated. Headache and vomiting were severe on August 15 and 16. Weakness of the limbs and photophobia were soon accompanied by respiratory difficulty. An airplane was sent in but could not land because of fog. During the night of August 16 his aunt continuously applied hot foments to his chest to aid his breathing, though she knew nothing of the experimental work which had been done on this problem at the Children's Memorial Hospital in Montreal. On August 17, the boy was flown to Montreal in a cyanosed condition. His C.S.F. showed 130 lymphocytes, and a positive Pandy reaction. (Proteins were 70 mgm. %.) He was transferred to the Alexandra Hospital for respirator care. His case illustrated the need for a portable respirator for use in aircraft and ambulances. In a short time such a respirator was made available in Montreal, to run on 110 volt A.C. or 6 volt D.C., as required.

CASE 61

Mrs. A.J., aged 20. This patient was a British war-bride who had two days previously landed in Halifax on the *Aquitania*. Twelve hours before admission she had an onset of severe headache, stiffness of the neck and back, with repeated vomiting. Her last bowel movement had been a week previously. Her C.S.F. showed 140 cells, 80% polymorphonuclears. (Protein 53 mgm. %.) Pandy positive.

The problem presented by this case was epidemiological, but the Diagnostic Clinic was asked to advise the transportation authorities whether the 40 other passengers in her train car, and those who were on the boat should be advised, quarantined, or if no action should be taken. The patient had obviously contracted the disease in England, where there was no epidemic, or on the *Aquitania*, possibly from a healthy carrier. The case is reminiscent of that of a healthy two-year old child shown by Kramer[4] to be a carrier of poliomyelitis virus, though there was no history of contact. Tonsil and adenoid tissue from the child produced poliomyelitis in monkeys as confirmed by repeated transmissions.

DISCUSSION

Before discussing the theoretical aspects of the disease itself, it might be well to sound a warning in the practical sphere. Some clinics have made a practice of doing lumbar punctures only in those cases showing no paralysis.[5] One death resulted in the Montreal epidemic from failure to perform a spinal puncture before sending a woman into a poliomyelitis ward for treatment. The patient showed what was taken to be "paralysis" and pain in the right leg. It turned out to be immobility due to a psoas abscess from a ruptured appendix. The abscess became so large, and the patient's temperature so high that some other diagnosis than poliomyelitis had to be made. Unfortunately it was made too late to save this case.

The differential diagnoses to be considered are legion, and should be on a printed sheet on the wall or desk of any poliomyelitis clinic. The obvious ones are: tuberculous meningitis; encephalitis; thrombophlebitis; botulism; scurvy; lead poisoning; Guillain-Barré syndrome; brucellosis; virus pneumonia; sub-arachnoid hæmorrhage; and ruptured intervertebral disc. Meals and Bower[1] saw in addition, in the 1930 epidemic in California: pyelitis; alcoholic neuritis; embolic phenomena in acute endocarditis; C.N.S. syphilis; brain abscess; and congenital muscular diseases. One case of apiol poisoning has been reported in the literature as poliomyelitis in pregnancy![2]

Perhaps the most striking thing about poliomyelitis from the diagnostic point of view is the high proportion of anomalies one encounters. Some of our cases had limb paralysis with no neck or back stiffness, and vice versa. One case had a temperature of 100.6° F. when he came to the clinic, five days after the onset of symptoms. He complained of neck stiffness, anorexia, vomiting and muscle pains. He showed unequal and decreased deep reflexes, and slight muscle weakness, yet his cell count was zero and his C.S.F. proteins 32 mgm. %.

In view of the inconstancy of signs and symptoms traditionally held to be essential in making a diagnosis of poliomyelitis, it would be best to concentrate on a few objective clinical findings such as altered deep reflexes, demonstrable muscle weakness, the C.S.F. cell count and the Pandy reaction. Laboratory assistance can be called on for estimation of C.S.F. proteins and for electroencephalograms and electromyograms in rare instances. Adamson[5] would rely on a "diagnostic triad" of spontaneous pain in the head or back, stiffness in the neck or back, and fever. Hamilton[6] has given a model report on the serious epidemic in 1937-38 in Sydney, Australia, in which he states that headache, back pain and weakness were of greatest diagnostic importance in his series of 250 cases. He made a special study of the spinal fluid at different stages of the disease. He saw 43 cases on the first or second day after onset of symptoms. In five cases no cells were visible in the C.S.F. These cases were repunctured two to three days later when the cellular increase typical of poliomyelitis was found. He concluded that "a single lumbar puncture performed at the height of the

illness was of no value as a diagnostic measure in 4.5% of cases". His figures for percentage of cases complaining of headache and showing muscle weakness correspond exactly with the Royal Victoria Hospital figures.

Drowsiness was a presenting symptom in 11% of the Sydney cases and 12.7% of the Manitoba cases reported by Adamson. This contrasts sharply with the finding of somnolence in a large proportion of the encephalitis cases in the concomitant Manitoba epidemic. In only one case was drowsiness marked in the Royal Victoria Hospital series. In this case no monocytes were found in the C.S.F., as might have been expected had the case been one of encephalitis.

The cellular reaction of the cerebrospinal fluid has long been a point of contention among diagnosticians. Trask and Harper,[7] in a series of 246 cases, reported 28 in which less than 8 cells per c.mm. were found along with a negative Pandy reaction, despite the fact that 5 of the 28 had paralysis. All cases were punctured within 10 days of the onset of symptoms. A repeated tap in another 4 of these cases gave a cellular increase which was not paralleled by any increase in symptoms. This is an important finding since it establishes beyond all doubt the fact that the cases were poliomyelitis in the first place, despite the low cell count. Still another 3 of the 28 cases had a remission of symptoms, which was later followed by a recurrence which resulted in an increased C.S.F. cell count.

Unfortunately Trask and Harper make no reference to spinal fluid protein estimations by other than Pandy's rough method. The work of Meals and Bower[1] which they quote in confirmation of their findings makes no reference to exact C.S.F. protein determinations but merely describes a "colloidal-benzoin test" which is said to have shown that 79% of their cases had a "typical" curve, with 8.6% being negative and 8.6% having a "tuberculous meningitis type" of curve.

The next chapter in the C.S.F. cells and protein story was written by Andelman[8] and his active group who studied the large 1945 epidemic in Chicago. They clear the air at the outset by stating: "It should be pointed out that 'subclinical' poliomyelitis is the ordinary form of the disease and should be termed poliomyelitis. This type represented about 79% of the cases; the meningitic type only 13%; the paralytic form only 6%; and the fatal type only 0.7%. Even of the paralytic group only 2.3% remain permanently paralyzed."

These workers contrasted 45 control children who were not poliomyelitis contacts but were suffering from such infectious diseases as pneumonia, pertussis and measles with 4 groups suffering from poliomyelitis, as follows:

	Av. C.S.F. protein mgm. %
Paralytic poliomyelitis (44 cases)	70
Meningitic poliomyelitis (17 cases)	63
Poliomyelitis contacts with sub-clinical poliomyelitis (19 cases)	55
Non-poliomyelitis febrile children with pneumonia, measles etc. (45 cases)	33
Recovering poliomyelitis 45 to 65 days after onset (12 cases)	27

Andelman concludes that with 55 mgm. % C.S.F. protein, sub-clinical poliomyelitis cannot be called a "mild" disease. He is in accord with Bridge,[9] who studied 471 cases and 1,123 contacts in the Buffalo epidemic, that no progress can be made in the prevention or control of the disease unless its protein manifestations are thoroughly studied. It is in this field that C.S.F. studies can contribute most. Small scale studies may contribute more than large scale, as shown by one of the best pieces of observation to be found in the vast literature on poliomyelitis. Visser[10] in the 1939 epidemic in Holland was the visiting physician to a Foundling Hospital where two children contracted poliomyelitis. At once admissions, discharges and visiting were stopped for two weeks. Lumbar puncture on all the inmates showed that all the remaining twelve children and a majority of the nursing staff had cellular and protein changes in the cerebrospinal fluid.

For some years it was thought that the most exact method of measuring the extent of the disease in an epidemic was by determining the specific antibody produced in contacts and subclinical cases. Brodie[11] in 1937 showed that the percentage of non-paralytic cases having antibody was the same as in normal healthy children. Burnet[12] in the 1937-1938 epidemic in Australia showed that 70 to 80% of normal healthy adults had polio. antibody circulating. He stated:

"In no case was there any significant change in antibody content resulting from exposure or infection a month or two previously. Further, five of the patients with clinical paralysis had possessed relatively large

HISTORY FORM

Date..... Name..... Age.....

Address..... Phone.....

Referred by Dr..... Final Diagnosis.....

Presenting complaints: Examiner:

Family history of polio?.....

Temp.	Pulse	Respir.	Blood Press.
ENQUIRY	+ = YES	O = NO	LEAVE NO BLANKS.
Date onset:	Raw water:	Raw milk:	Polio contacts:
Onset fever:	Sore throat:	Anorexia:	Nausea:
Vomiting:	Diarrhœa:	Constipated:	Incontinent:
Headache:	Neck stiff:	Back stiff:	Muscle pain:
Dysphagia:	Voice change:	Dentistry:	Tonsillectomy:

PHYSICAL EXAMINATION	NERVOUS SYSTEM	MUSCLE WEAKNESS
	Reflexes: R. L.	R. L.
General:		
ENT:		Arm flexors
	Biceps	Arm extensors
Mucus	Triceps	Leg flexors
Cyanosis	K.J.	Leg extensors
Resp.	A.J.	Neck
		Intercostals
Heart	Others:	Others:
Abdomen		
	Skin irritability:	Spasm:
G.U.	Fasciculation:	Atrophy:

CRANIAL N. WEAKNESS: LUMBAR PUNCTURE:

Optic Fundi	co-operation:	F.P.....mm.	C.S.F. Wass.
3.4.6.	I.P.....mm.	Lymphs.	
	Polys.		Culture:
5. 7.		Protein.	
	Pandy.		
9. 10.		Sugar.	EMG ordered:
	Chlorides.		
11. 12.			

REMARKS:

amounts of antibody in their blood at the time of their acute illness, and equally therefore at the time of their infection a few days before''.

With this evidence the use of serum was fairly well abandoned and the former reliance on neutralization tests was tempered considerably. Burnet concluded that: ''Previous infection with an antibody-producing strain does not ensure immunity from paralysis by an epidemic strain''.

Burnet's work is of great importance in understanding the possibly changing nature of the disease. As Sweden, the United States, New Zealand, and Australia have reached a high standard of living and a low death rate they have simultaneously built up the worst poliomyelitis histories in the world. Burnet asks if what was once a pharyngeal disease of children is being driven into hiding in the central nervous system of adults. The 19th century virus was epitheliotropic, carried by adults, antibody-producing, and through recurrent asymptomatic episodes produced eventual immunity. The 20th century virus is neurotropic, spread by children, and incapable of producing more than a temporary immunity. Recurrent infections in one individual and multiple infections in one family bear out this hypothesis.

CONCLUSIONS

1. The clinical diagnosis of poliomyelitis, in the experience of the Royal Victoria Hospital Diagnostic Clinic in the 1946 epidemic, was most reliably made from: (*a*) A history of stiffness or soreness of the back, usually with pains in the muscles of the body. (*b*) Diminished or unequal reflexes. (*c*) Demonstrable muscle weakness. (*d*) Increased cells and protein in the cerebrospinal fluid. The diagnosis could be made even when some of these findings were missing.

2. In this series of cases, age, temperature, a history of headache, anorexia, nausea or vomiting, or sore throat failed to distinguish poliomyelitis from other concurrent diseases. The interpretation of Kernig's sign by the clinic staff has been found to be too variable to make it of certain diagnostic value.

SUMMARY AND RECOMMENDATIONS

It can be said that the 1946 epidemic in Montreal emphasized the need for greater preparedness in facilities for diagnosing and studying the disease. Completeness of examination and of records could best be assured through the use of a printed form such as the questionnaire herewith. It has probably many shortcomings but it is an attempt to provide something in our clinic where nothing existed before. The literature is strangely inadequate on this matter, though a mixed epidemiological and diagnostic questionnaire was recently published by the group which covered the 1945 epidemic in Mauritius so completely.[13] Doubt may arise in some minds as to the value of questioning suspects concerning recent tonsillectomy or dental work, but it is felt that the unequivocal observations of Stillerman and Fisher[14] and the experimental work of Sabin[15] on this problem make it essential to record this information routinely.

Next after records in importance come methods of examining the C.S.F. Differential cell counts using a trichrome dye are needed. The Pandy test could well be replaced with an accurate determination of the proteins in a photoelectric colorimeter in the clinic proper, using Cipriani and Brophy's[16] simple and rapid method.

Finally, portable equipment for artificial respiration is required for the safe transport of diagnosed cases to infectious disease hospitals.

Acknowledgment is made of the assistance rendered by Dr. Donald McEachern and Dr. A. W. Young as consultants to the clinic, by the following members of the resident staff, Dr. Dustan, Dr. T. F. Rose, Dr. N. Sloan, Dr. H. T. McAlpine, Dr. K. Fleming, Dr. R. Rabinovitch, Dr. B. F. Graham and by the R.V.H. Nursing Staff.

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RÉSUMÉ

Le diagnostic de la poliomyélite doit se baser sur les faits suivants: une histoire de raideur ou de douleurs vertébrales habituellement accompagnées de douleurs musculaires; la diminution ou l'inégalité des réflexes; une parésie notable et une augmentation des éléments et des protéines du l.c.r. Le diagnostic peut aussi se poser sans que ces signes soient au complet. On devrait être plus prêt à faire face à une épidémie. Un questionnaire clinique et épidémiologique est une aide précieuse. L'examen du l.c.r. est essentiel et on fera bien de mesurer le degré de l'albuminorachie.

JEAN SAUCIER.

CARCINOMA OF THE LEFT COLON*

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IN the past ten years, the operability rate for carcinoma of the colon has increased, the operative mortality has diminished, and the five-year survival rate has greatly improved. These features are most gratifying, but it is still disappointing to find that the interval between onset of symptoms and the final diagnosis has been relatively only slightly affected.

The diagnosis may be difficult and there are no short-cuts. A reliable and careful history, a thorough examination, and a knowledge that such a lesion does occur constitute the first line in the battle. The simple office procedure of a digital examination and a sigmoidoscopic inspection are invaluable, with x-ray and biopsy procedures as final confirmation. Let me emphasize that no patient should be operated upon for hæmorrhoids without a sigmoidoscopic examination as a preliminary.

TABLE I.

DISTRIBUTION OF MALIGNANT LESIONS OF THE LARGE BOWEL

	%
Anus, rectum and recto-sigmoid	62
Sigmoid, descending colon and splenic flexure	20
Transverse colon	5
Hepatic flexure, ascending colon and cæcum	13

From Table I, showing the distribution of malignant lesions of the large bowel, it is apparent that 60% of the cases are palpable on digital examination and an additional 10 to 15% are visible through a sigmoidoscope. The disease affects both sexes in approximately equal proportions and no age is exempt. I recently had a carcinoma in a child of fourteen.

CASE 1

I.M., aged 34. This female patient presented herself in September of 1946 with a history of bleeding per rectum at intervals for four years. She had had intermittent bouts of constipation accompanied by crampy, abdominal pain. Otherwise, she was in perfect health and the review of systems and of past illnesses was of no significance. On examination, she was a well-nourished, rather nervous woman. General physical examination was negative. Rectal examination showed no evidence of fissure or hæmorrhoids. Proctoscopic and sigmoidoscopic examinations were negative. A barium enema (see Fig. 1) revealed a temporary obstruction in the region of the junction of the descending and sigmoid colon but the barium flowed past the area freely and completely filled the colon. Multiple studies of the suspicious area were made and demonstrated a filling defect. The radiologist was of the opinion that there was a pedunculated polypoid lesion causing the obstruction. At operation, a large pedunculated lesion was found with an extensive infiltration of the bowel wall at the base of the polyp. A resection with end-to-end anastomosis was done and the patient made an uneventful recovery. The pathological report was returned "adenocarcinoma".

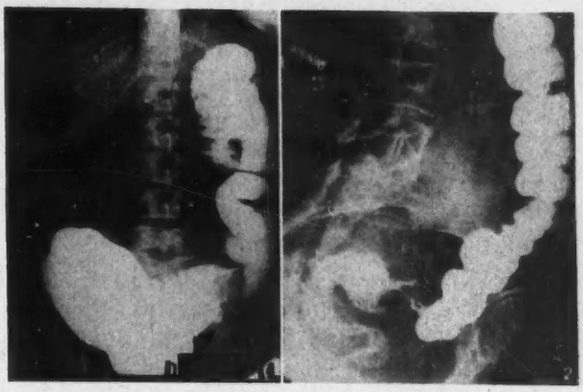


Fig. 1.—I.M., aged 34. Barium enema showing filling defect at the junction of the descending and sigmoid colon. **Fig. 2.**—E.H.L., aged 64. Barium enema April, 1946, showing a large filling defect in the sigmoid colon.

CASE 2

E.H.L., aged 64. This patient had been attended by an osteopath for some time. She was seen in January of 1945 complaining of streaks of blood at stool. There was no loss of weight; bowels regular, no mucus, diarrhœa or constipation. General physical examination was entirely negative as were pelvic and rectal examinations. Proctoscopic and sigmoidoscopic inspection revealed no disease in the terminal large bowel. A barium enema was given and all parts of the colon were well-outlined and appeared normal. The only positive finding at that time was a secondary anæmia. The red blood count was 3,000,000 with a hæmoglobin of 65%. Nine months later she returned having lost 20 pounds in the previous four months. Her appetite was good and she had no constipation or diarrhœa. She had frequent lower abdominal pains with a desire to defæcate. Rectal examination was negative. In April of 1946, she returned again complaining of crampy abdominal pain and weakness. Sigmoidoscopic examination was negative. A repeated barium enema (see Fig. 2) showed a filling defect in the sigmoid colon. At operation, an annular constricting lesion was found which showed no evidence of metastases. A wide resection was done with an end-to-end anastomosis. She made an uneventful recovery. The pathological report indicated an adenocarcinoma.

CASE 3

A.N., aged 50. This patient was first seen in 1933. Her complaint at that time was passing dark clots at stool for many months. In 1929 she had had a fissure-in-ano and in 1930 a hæmorrhoidectomy. There was no significant history other than the fact that she had been constipated all her life. Physical examination was entirely negative and a barium enema showed a normal large bowel. Two years later, she reported again with more bleeding. Physical examination and barium enema were repeated and again were negative. In February of 1947, she reported again complaining of crampy pain in the left lower quadrant and bleeding consistently at stool for three weeks. A barium enema was attempted but a complete obstruction was encountered in the upper sigmoid colon. Operation was advised and revealed an annular constricting carcinoma of the sigmoid colon.

* Read at the Seventy-eighth Annual Meeting of the Canadian Medical Association, Section of Surgery, Winnipeg, June 26, 1947.

A wide resection was done in a modified Mikulicz's manner. Pathological report was "adenocarcinoma of the large bowel".

These three cases illustrate the difficulty of demonstrating a lesion which is producing bleeding per rectum and the value of repeated examinations.

SURGICAL MANAGEMENT

Preoperative preparation.—The improvement in mortality and survival of recent years is the direct result of improved preparation and surgical management. A large portion of the credit has been ascribed to chemotherapy, but I think undoubtedly it is only one factor and probably a relatively small factor in the direct improvement in results. Sulfathaladine or sulfasuxadine is given for four or five days preoperatively and during this time other details of the preparation can be thoroughly completed. The patient is put on a low-residue diet; daily colonic irrigations; the fluid balance is stabilized; blood grouping is determined; anæmia is corrected; the blood protein level is assessed as well as complete blood chemistry, renal function, etc. An adequate period of time is allowed to prepare the patient in all respects.

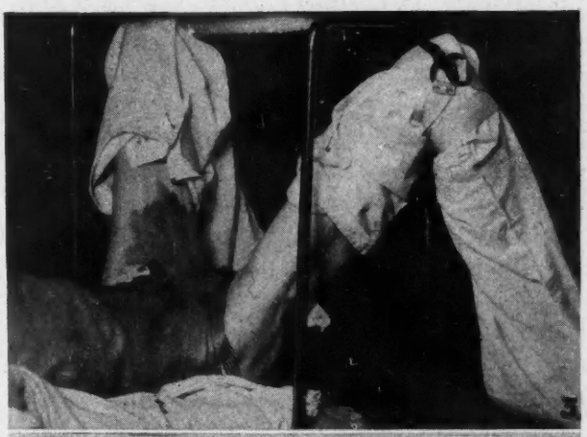
Anæsthesia.—The preoperative sedation consists of seconal gr. 1½ one hour and a half before operation and morphine gr. 1/6 with hyoscine gr. 1/150 three-quarters of an hour preoperatively. If the patient is in first-class condition, the anæsthetic of choice is pontocaine and glucose with a very small amount of pentothal for sedation. In the majority of cases, the patients are older and not first-class risks and our anæsthetists prefer pentothal induction followed by cyclopropane and curare. One thousand c.c. of blood is valuable routinely and it is found that as a rule it is desirable to give 500 c.c. rather rapidly during operation.

Selection of operation.—The fundamental principles underlying the surgical treatment of carcinoma of the colon and rectum have not changed in the past few years but the application of various methods and procedures has been constantly modified. The one paramount principle which applies to all cases of malignant disease, no matter where the location, is that there is only one opportunity to do a complete radical ablation of the disease. Opportunity never knocks twice. This means a wide resection of the lesion including its gland-bearing area and to accomplish this, it is difficult to apply a single procedure in all cases. The technical difficulties in individual cases must be overcome by varying and accommodating the operative details to suit the case. In lesions from the transverse colon to about the recto-sigmoid junction or just above it, my own preference where possible, and it almost always is possible, is to do a resection with an immediate end-to-end anastomosis by an open or closed method. Postoperative peritonitis in large bowel surgery is due not to contamination or soiling at the time of operation but to leaking suture line. With carefully placed interrupted fine silk sutures, the incidence of postoperative peritonitis compares favourably with that complication following Mikulicz's.

If the patient presents an obstruction when first seen, preliminary decompression is essential to success. If the patient is greatly distended and tense, a simple cæcostomy may be all that the patient will stand. However, it is not a very satisfactory method of preparing the bowel for resection and I find that it is necessary to do a colostomy a few days later. If a colostomy is done, it should never be done in close proximity to the lesion itself but at a distant point so leaving the field about the lesion completely free from previous operative reaction and adhesions. In some instances where the obstruction is of recent occurrence and the distension is not great, there is still a place for a Mikulicz's type of resection but the disadvantages of this operation are rendering it less and less frequently indicated. The prolonged hospitalization, multiple operations, infected wounds, and weak scars are the price demanded in the Mikulicz's procedure.

In lesions about the peritoneal reflection at the recto-sigmoid junction, the technical difficulty of an end-to-end anastomosis is very great. In the hands of the average surgeon, it is associated with a much higher mortality and rate of recurrence. In my opinion, the preservation of the sphincter should never influence one's judgment or decision in preference to radical resection. I think in the vast majority of cases, the prejudice against a colostomy in the minds of laymen and perhaps of the physician too, is based on the desperate state and experience of the patient in whom the lesion is not resectable. The unpleasant, disagreeable features are the result of the lesion and not the colostomy.

In our clinic, we do abdomino-perineal resection in a truly combined manner with simultaneous dissection of the perineal and the pelvic portion. The patient is placed in a modified lithotomy position (see Fig. 3) with the knees abducted, the hips flexed to less than a right angle and the patient draped so that the abdominal and perineal fields are both accessible. A midline, lower abdominal incision is made and the abdomen explored to determine whether or not the case is operable. If no metastases are found in the liver or distant lymph nodes, then while the dissection from above proceeds, a surgeon commences the perineal dissection and the two dissections meet in the hollow of the sacrum (Fig. 4). This



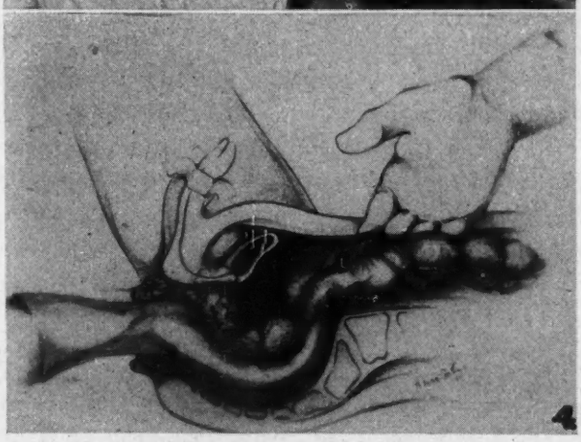


Fig. 3.—Illustrates the position for combined simultaneous abdomino-perineal resection. **Fig. 4.**—Diagram illustrating the simultaneous dissection from above and below in combined abdomino-perineal resection.

method of approach was first demonstrated to me by Mr. Rodney Maingot, of London, England. It reduces the operation to a remarkably easy procedure; there is no change in position during operation; shock is tremendously reduced; and, though of less importance, the operative time is less than one hour. The colostomy is brought out through a left inguinal incision and the redundant bowel amputated. The perineal incision is partially closed with a few interrupted stitches and a cigarette drain is left in the hollow of the sacrum.

The postoperative care is not elaborate. The clamp is removed from the colostomy at the end of thirty-six or forty-eight hours and the cigarette drain in the perineum is removed at the same time. The patient is kept on liquids for three or four days followed by a soft diet, with liquid paraffin twice daily. As the colostomy commences to function, the diet is increased and the liquid paraffin reduced. The patient is out of bed on the fourth or fifth day and is usually able to go home at the end of three weeks.

Colostomy management.—The management of the colostomy is not at all difficult and need not make a social outcast of any patient. During the period of hospitalization, the colostomy may require irrigating every other day but in the majority of cases by the time they are discharged from hospital this problem has resolved itself to an unnecessary practice. Occasionally, patients will cling to the ritual as if their life depended on it and it is well to allow them to satisfy their desire. If they do persist with irrigations, it is advisable that they be done in the evening so that if there is some delay in complete emptying they can wear a large pad on retiring and not soil their clothing. During the day, they wear a small pad of gauze covered with a piece of oiled silk and just an ordinary canvas belt or binder. We have found colostomy bags and contraptions rather undesirable and uncomfortable to wear. They are unsightly underneath the clothing and very unpleasant to clean. In our experience, patients provided with these devices discard them of their own volition at the end of a few weeks and resort to a simple pad and binder. After a short period of trial and error, we find the patients are able to regulate their diet by themselves with very little advice and after a few months the frequency of irrigation is reduced.

A summary of our cases since resuming civilian practice is as follows:

	Cases
Transverse colon	6
Splenic flexure	1
Descending colon	3
Sigmoid colon	16
Rectum	14
Total	40

Of these, 10 cases were found to be inoperable at laparotomy. Of the 30 remaining, there were 4 deaths from operation, an average of 13%. The average age was 57. The range was from 34 to 76. Of the 30 cases resected, 14 were abdomino-perineal resections with one death, one Mikulicz's obstructive resection was done and the remaining 15 cases were resections with end-to-end anastomosis.

SUMMARY

A brief discussion is presented on the diagnosis of carcinoma of the left colon with three case histories illustrating the value of repeated examination. Preoperative preparations, choice of anæsthetic and selection of operation are presented. A new method of carrying out combined abdomino-perineal resection of the rectum is described. The problem of a colostomy is resolved to a simple management.

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RENAL COMPLICATIONS IN YOUNG DIABETICS*

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DURING the past 25 years great advances have been made in the understanding of diabetes mellitus, with consequent improvement in the treatment of patients suffering from this disease. Early in this era it became apparent that diabetes mellitus must be divided into adult and juvenile. As time went on the older diabetics naturally died off first. Abundant statistics are now available concerning the incidence of arteriosclerosis, infections, cataracts, cardio-vascular and other degenerative diseases which eventually contribute to the cause of death in this older group.[1] In 1936, rumblings occurred when Kimmelstiel and Wilson[2] reported a new entity called intercapillary glomerulosclerosis, which they found in elderly diabetics. Their youngest patient was 51 years of age. In 1939, Newburger and Peters,[3] and in 1941, Porter and Walker[4] and others[5, 6, 7] reported intercapillary glomerulosclerosis as a clinical syndrome occurring in diabetics. The ages of all the reported cases varied from 41 to 69 years and averaged 58 years.

During these 25 years at the Montreal General Hospital, under the direction of Dr. I. M. Rabinowitch, and more latterly Drs. Fowler and Bensley, a large diabetic outdoor clinic was established. When these clinic patients showed any signs or symptoms of not being well they were immediately referred into the hospital for investigation. Arrangements were made with the hospital that, while the Metabolism Department was responsible for the diabetic management, the patient's general condition was the responsibility of the attending ward physician.

We were watching the progress of the young diabetic. He or she attained normal growth, did reasonably well in school, college, and later at work. It was generally felt that they would live a healthy life with normal expectancy. Our first jolt came in 1941. A young diabetic patient, aged 30, was admitted to hospital and died in uræmia. Since then we have had four similar deaths in this age group, with complete post mortem studies. Furthermore, in the outdoor clinic are now seven young diabetic patients already showing signs of this same syndrome.

It is the purpose of this communication to point out the occurrence of renal complications in an alarmingly high proportion of young diabetics.

SUMMARY OF MAIN FINDINGS IN FIVE JUVENILE DIABETICS

CASE SEX	AGE DIABETES ONSET	AGE DEATH	DURATION DIABETES YEARS	AGE COMPLICATIONS ONSET	DURATION SYNDROME YEARS
1 (F)	12	30	18	28	1.5
2 (m)	4	18	14	17	2.0
3 (F)	11	23	12	20	3.0
4 (F)	23	37	14	36	1.5
5 (m)	17	32	15	30	1.5
AVERAGE	13.4	28	14.6	26.2	1.9

Chart 1

* From the Department of Medicine, the Montreal General Hospital. Presented at the Annual Meeting of The Royal College of Physicians and Surgeons of Canada, November 15, 1946.

Chart 1 summarizes the main features of the present series, comprising our first five fatal cases. Three of the five were female. The age of onset of the diabetes varied from 4 to 23 years, averaging 13.4 years. The age of death averaged 28 years. The total duration of diabetes averaged 14.6 years. The average age at which complications were detected was 26.2 years. The average duration of life after the onset of renal complications was 1.9 years.

Etiology of Renal Complications

The cause of this complication is not known. All five patients had several hospital admissions. On reviewing the case histories, the following contributing factors were common to all.

1. Duration of the diabetes.—An average of 12 years elapsed before symptoms or signs developed. The control of the diabetes was difficult.

2. Diet.—In retrospect, the diets prescribed during the early insulin era were wholly inadequate for growing children. Furthermore, by calculation it could be determined that these patients for the most part were in negative nitrogen balance.

3. Breaking the diet.—This was a frequent occurrence and on several occasions necessitated admission to hospital.

4. Repeated infections.—Frequent head colds, sore throats, the common childhood diseases, all produced a strain on the diabetic control. Two patients suffered a severe bout of "catarrhal jaundice", precipitating coma.

5. Frequent reactions.—The blood sugar levels were extremely labile. While only a few cases exhibited bouts of coma, insulin reactions were a frequent and distressing occurrence.

Diagnosis and Clinical Course of Renal Complications

The onset of the renal complications was insidious. Albuminuria, as a slight trace, was an early and constant finding. A careful microscopic examination of the urinary sediment would show an occasional red blood cell. The blood pressure would be variable, but could be found at some time to be elevated above the normal for the age. The blood urea nitrogen values were normal, but always at the upper limits of normal. The ocular fundi and the heart appeared normal. These findings may occur five to six years or more before the clinical picture becomes definitely manifest.

Then, after the duration of twelve years on the average, the following changes appeared rapidly.

1. The urine showed albumin consistently in large amounts, graded as one plus or more, with a few red blood cells and a few casts.

2. The blood pressure was increased, fluctuating widely, it is true, but with a more or less persistent elevation of the diastolic pressure.

3. Retinopathy leading to blindness. This was an outstanding feature. A sudden onset of impaired vision, involving one eye, was a common symptom. "A dull heavy feeling" over the eyebrow and pain behind the eyeballs were universal complaints. In two patients the pain, uncontrollable by opiates, necessitated retrobulbar injection of local anæsthetic for relief and, later, enucleation of the eye because of infection. Marked arteriolar changes were present but the prominent finding was massive hæmorrhage with huge extravasations lifting portions of the retina, leading to great distortion. So universal were the eye symptoms and findings that the progress of the syndrome could be judged by the extent and progress of these changes. Indeed, two of our recent patients sought medical advice because of eye symptoms alone. One of them was admitted to the service of ophthalmology. In all cases, the findings of a retinopathy heralds the onset of the final stage.

4. Gastro-intestinal complaints. These individuals were troubled with indigestion. They suffered bouts of nausea and vomiting. Loss of appetite, alternating diarrhœa and constipation, discomfort, rumbling of gas, were common symptoms.

5. Œdema, generalized in distribution, was present in all. Puffiness of the face, swelling of the ankles, occurred from time to time. Hypoproteinæmia was present.

6. Anæmia appeared late and was usually associated with nitrogen retention.

7. The blood urea nitrogen and creatinine levels rose, leading to death in uræmia approximately two years from the onset of the final stage.

Post Mortem Findings*

The kidneys at autopsy in all cases were not

* As reported by the late Dr. Lawrence Rhea, and Drs. J. Pritchard and W. Matthews.

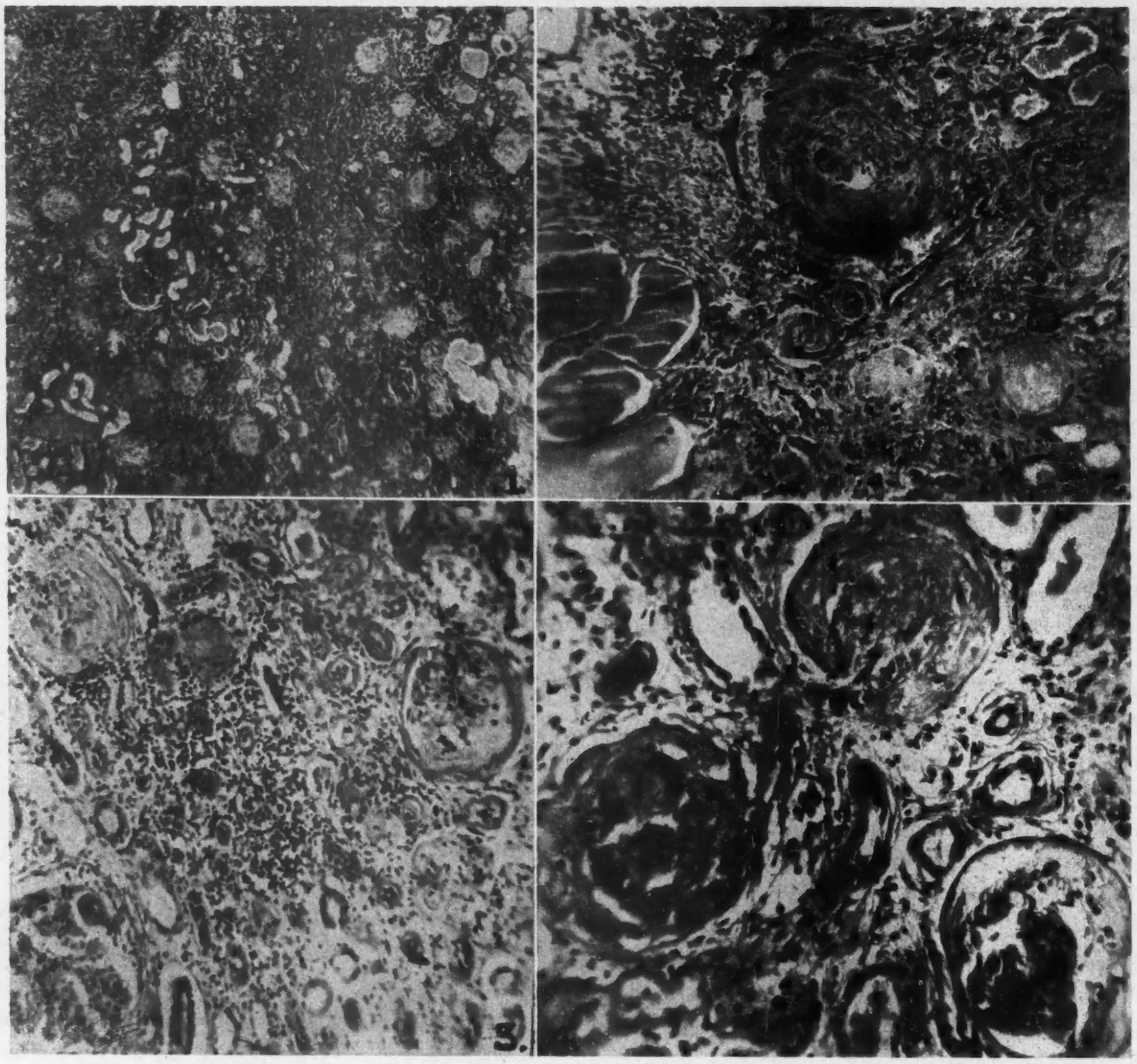


Fig. 1.—Widespread involvement of kidney parenchyma, with glomerular sclerosis, tubular atrophy and dilatation. The renal capsule is thin. (x 60). **Fig. 2.**—A fibrosed glomerulus with atrophic shrinkage of many tubules and dilation of a few containing albuminous casts. Two arterioles are sclerotic and there are foci of chronic inflammatory cells. (x 150). **Fig. 3.**—Varying degrees of glomerular sclerosis and widespread tubular atrophy with interstitial lymphocytic exudate. (x 150). **Fig. 4.**—Higher magnification illustrating similar changes. (x 400).

grossly contracted. Microscopically they showed (Figs. 1 to 4) a mixed lesion with diffuse glomerular sclerosis and hyalinization, diffuse tubular atrophy and interstitial lymphocytic infiltration of the pyelonephritic type, together with mild to moderate intra-renal vascular lesions in the nature of hyaline arteriolar sclerosis and intimal thickening of larger arteries. The focal hyaline glomerular deposits of Kimmelsteil and Wilson were not seen.

TREATMENT

We are profoundly ignorant of the true nature and cause of these severe renal lesions occurring in youthful diabetics. Until more is known, all efforts should be devoted to prevent the occurrence of this renal syndrome. Treatment during the first few years of the disease is important. We must aim at ideal control of the diabetes. By this is meant better control of the diet, adequate to maintain nitrogen balance, control of infections, regulation of activity, and prevention of wide blood sugar fluctuations.

CONCLUSION

This is a report of the clinical and pathological findings in five cases of diabetes mellitus in which the onset was at an early age. They all terminated fatally within an average of 14.6 years with renal disease. The kidney lesion appears as a combined inflammatory and hyaline degenerative vascular one, and it leads to

renal failure, being associated with albuminuria, hypertension, œdema, hypoproteinæmia, nitrogen retention, anæmia, and generalized degenerative vascular lesions which are particularly severe in the eye, progressing rapidly to blindness, in each of the five cases.

The treatment[8] is unsatisfactory and it is hoped that future studies will throw some light on the cause and mechanism of renal complications in diabetes, particularly the young diabetic.

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MARROW BIOPSY AND ITS PROCESSING FOR HISTOLOGICAL PREPARATIONS*

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THE stimulating reports by E. M. Schleicher of the Minneapolis General Hospital on Miliary Tuberculosis of the Bone Marrow Organ[1, 2] have provided the impetus for certain bone marrow studies presently nearing completion at the laboratories here. The nature of these investigations has demonstrated the importance of a reliable standard technique for the acquisition and preparation of bone marrow for histological study. Such preparations, preserving as they do the tissue architecture, stand apart from the uniquely useful though distorted cytological picture presented by the hæmatologist's smear.

The following method, used at the Mountain Sanatorium, while in no wise an original technique in its entirety, has been found sufficiently gratifying in its constant presentation of acceptable specimens to warrant its being brought to the attention of co-workers in this field.

The steps followed, with the exception of some minor, though in our opinion important modifications, are in effect, essentially the same as those traced in an article by Barbara Tucker, B.S., M.T.[3] of the North Western Hospital, Minneapolis, and enclosed in a personal communication from Dr. Schleicher on the same subject. The modified technique outlined below has provided us with paraffin sections of constant quality in some 200 consecutive sternal aspirations, and, while it is neither our purpose nor intention here to epitomize those conditions in which sternal marrow aspirations are of value, we regard the technique as capable of providing specimens of biopsy calibre while maintaining its place, from the patient's viewpoint, of a minor procedure.

The needle used (Fig. 1) is considered suitable in its possession of an adjustable guard and the ease with which it may be taken apart for purposes of thorough sterilization. Although the actual site chosen for aspiration (Fig. 2) we have found to be not of paramount importance, the area of the sternum opposite the second interspace is usually selected if for no other reason than that the surface markings of related structures (*e.g.*, angle of Louis) make it more easily defined. The thoroughness of skin preparation and the degree of surgical asepsis used should conform at least to the minimum associated with all such procedures. We have found the pneumothorax towel with its central opening a very useful adjunct to other accessories.

The introduction of a local anæsthetic (novocaine 1%) is carried out in the manner common to all such procedures, the infiltration being carried finally down to and through the periosteum. Gentle massage with a swab and the lapse of two minutes make the anæsthetic more effective. The needle, with the stylet *in situ*, is introduced through the skin which is at that time placed under tension between the thumb and index finger of the opposite hand. It is introduced with steady pressure and a continual rotary movement beginning from the moment the needle point has pierced the skin surface. In this manner there is no sudden sensation of collapse appreciated by the patient as the anterior table is pierced, while the decreased resistance is easily communicated to the operator's hand. The patient's only sensation is one of pressure unassociated with pain.

Once the needle is in position the stylet is removed and a 75 c.c. syringe with adaptor is

* From the Laboratories of the Mountain Sanatorium, Hamilton, Ontario.

firmly attached, some little attention being paid to this last point. The plunger of the syringe is then withdrawn sharply to a point as high as the negative pressure will allow (usually to the 20 or 30 c.c. mark). Although this causes a disquieting, but momentary, sensation of "rushing" in the patient's chest we are convinced that it is an extremely important manœuvre in that the sudden high negative pressure thus produced is an important factor in breaking away a sufficiently large quantity of marrow particles for the making of satisfactory sections.

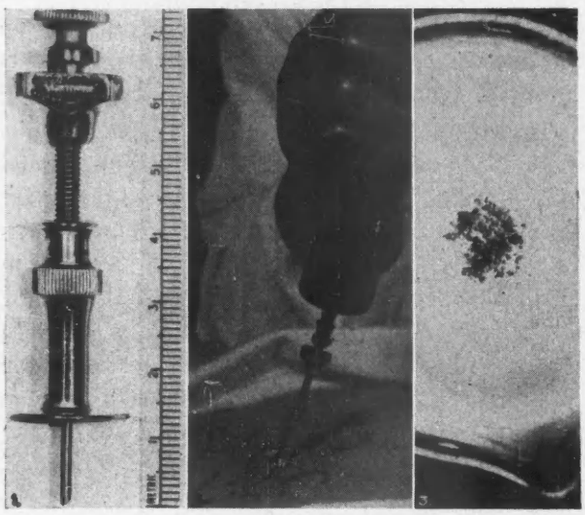
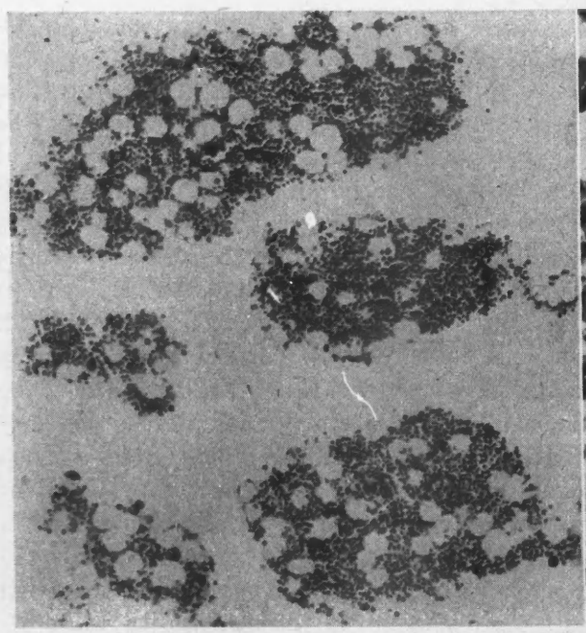


Fig. 1.—Sternal aspiration needle. (Manufactured by V. Mueller & Co., Ogden Avenue and Van Buren St., Chicago, Ill.) **Fig. 2.**—An aspiration in progress. **Fig. 3.**—Aspirated "units" lying in No. 100 mesh wire screen during washing process.

While the quantity of tissue removed will vary in proportion to the demands to be met, we have found that a quantity of 5 to 6 c.c. usually contains a sufficiently large number of marrow particles to permit of serial sections through a representative part of the tissue. Once this has been obtained the syringe may be gently detached and its contents transferred forthwith to a container, usually a test tube, with waxed interior, containing approximately 3 mgm. of heparin. It is then agitated gently, after which it may be permitted to stand for several hours without any deleterious effect if refrigerated.

While in the past attempts have been made to transfer "units" to the fixing solution by utilizing pipettes, eye droppers, etc., it has been found expeditious and completely effective to transfer the contents of the tube to a beaker and to separate the marrow particles by passing the whole through No. 100 mesh wire screen (Fig. 3), when the units remain on the proximal side of the mesh. These may be thoroughly washed with fixing solution before being transferred to a container, by the simple method of inverting the screen and washing the whole into a small beaker by pouring a further quantity of the solution through the reverse side. This procedure appears to obviate the



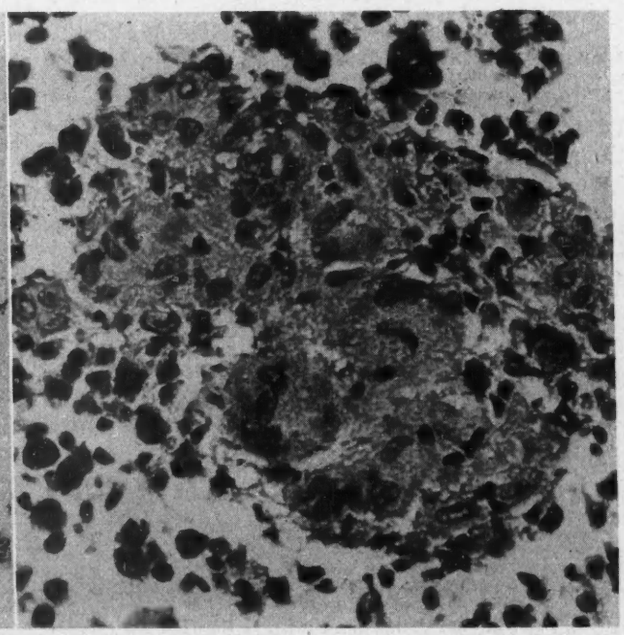


Fig. 4.—Marrow "units", very low power, H & E stain. **Fig. 5.**—Marrow "unit" showing typical epithelioid tubercle with early giant cell formation, high power. H & E stain.

necessity of repeated renewal of the fixing solution (10% neutral reagent grade formalin in normal saline) as is otherwise essential to free the tissue of the contaminating red cells.

At the end of the hour required for satisfactory fixation the process of dehydration is carried out by successively transferring the "units" to 50, 75, 85 and 95% alcohol. The transfer is effected on each occasion by pouring the suspended marrow particles on to the screen and utilizing the next solution as a means of washing them back into their original container. Thirty minute intervals were found to be sufficient for each of the alcohols mentioned above.

The marrow is subsequently immersed in absolute alcohol for 15 minutes and the dehydration completed by final transfer for a further 15 minute interval to 1 to 4 dioxane. The low specific gravity of the latter permits the marrow to settle to the bottom of the container and the dioxane may, with relative ease, be decanted or aspirated.

Clean paraffin, at a temperature of 58° C. and in quantities of from 30 to 40 c.c. is poured directly into the beakers containing the marrow and the whole incubated at this temperature for a period of not less than one hour and not more than two. After this interval the paraffin in excess of 3 to 4 c.c. is decanted. The remainder is gently agitated and, while the marrow is still in suspension, the contents poured directly into a small paper boat. Any remaining particles may be washed out with warm paraffin, utilizing a warmed pipette for the purpose.

In this manner there is no direct handling of the tissue and, by utilizing a boat approximately 1 cm. square, there appears to be no necessity for subsequent orientation of the particles in the paraffin.

Following cooling, the cutting and H & E staining of the sections are carried out in the usual fashion.

The quality of the preparations obtained with the foregoing technique is illustrated in Figs. 4 and 5.

The authors wish to acknowledge the skilful technical assistance of Miss S. G. Tonner. They also wish to express their indebtedness to Dr. C. H. Playfair, Medical Superintendent, for permission to publish this note.

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CONGENITAL CYSTIC DISEASE OF THE LUNG*

(A Familial Incidence of Four Males in One Family)

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Ninette, Man.

Schenck,[1] Sellors,[2] and Willis and Almeyda[3] have published excellent studies of congenital cystic disease of the lung. Willis and Almeyda, the most recent of the three, state that no cases illustrating the familial incidence of the disease have appeared in medical literature. The four cases reported here occurred in a French-Canadian family of eleven children. Careful investigation into family history revealed nothing that might be considered to have any bearing on a familial condition.

Case 1

Male, aged 20 years. Had worked as plumber's assistant from age 14 up to time of his final illness. There is no history of early illness except that he had had a discharging left ear from childhood. At 14 he had an illness diagnosed as "pleuro-pneumonia". He made a seemingly good recovery but from that time on was more subject to colds. He was knocked down and dragged by a car at 17 but claimed that he suffered no injury. A year later he took a worse-than-usual cold at Christmas time and it was this that led to his examination at the tuberculosis clinic on February 24, 1932 when a diagnosis of far advanced tuberculosis was made and he was admitted to the sanatorium: the clinic notes are as follows: "dry cough since early winter; no sputum; no pain; has dropped four pounds; dyspnœa is evident; has marked dorso-lumbar scoliosis but no kyphosis; weight 102 pounds and is very thin; cheeks flushed (cyanotic?); dulness in upper half both lungs; crepitations in LL". In sanatorium, May 5, 1932 to September 4, 1933, he was without symptoms, tuberculin was negative and he gained to 112 pounds, his best weight. After return home he followed rest routine but took ill suddenly and died after a night of marked dyspnœa, cyanosis and spitting of blood. Autopsy was not done.

Comment.—The sudden and violent onset of this terminal episode suggests spontaneous pneumothorax; his eldest sister, who was with him during the night, thought he vomited the blood, but the marked dyspnœa and extreme cyanosis that were not relieved appear to indicate that the source of bleeding was pulmonary; on the other hand spontaneous rupture is unlikely to cause hæmoptysis. Note in the pathologist's report on Case 4 that, "intra-alveolar hæmorrhage" was present; it seems probable that the terminal episodes in these two cases were the

* Compiled from the records of the Sanatorium Board of Manitoba under the supervision of Dr. E. L. Ross, Medical Director.

same with the recurrent colds in Case 1 corresponding to the asthmatic spells in Case 4.

Case 2

Male, aged 12. He is said to have had measles, mumps, whooping cough, scarlet fever and chicken pox, but we cannot be sure. At any rate, he was doing poorly and was brought to the clinic August 12, 1933, when 10 years old; here is the report: "Weight 55 pounds; thin and pale; is losing weight and looks miserable; does not play around with the other children; coughs in the morning since last summer; gets repeated colds; many carious teeth. Chest film shows fine infiltration generalized throughout both lungs with slightly more in the left; there are fine crepitations at both roots."

Diagnosis of tuberculosis was made and treatment advised. He was in sanatorium September 14, 1933 to May 13, 1934 without symptoms; tuberculin reaction was "persistently negative"; he gained three pounds. He spent the summer of 1934 at home going to school, but by July cough had recurred, weight was dropping and he looked poorly so was re-admitted October 23, 1934 to July 11, 1935. At this second admission his film showed spontaneous pneumothorax on the left, but temperature was normal, sedimentation 7 mm. (Cutler) in an hour, gastric contents negative for tubercle bacilli; response to human type old tuberculin negative; white cell count 10,000. In January 1935, a test with 1/1,000 avian tuberculin was 3 plus. Tonsillectomy was done under ether. In March he began to run fever, 100 to 101°, and this continued until death. In April left pneumothorax was started but spontaneous pneumothorax occurred with resultant hydrothorax so refills were abandoned early in June. He became worse and died July 11, 1935. Autopsy report was as follows: "Fibrocaseous tuberculosis of both lungs, particularly advanced in left apical area; numerous adhesions and a large subpleural hæmorrhage in left lung; there are also petechial hæmorrhages in heart, kidneys, stomach, bowel and omentum; it cannot be stated whether disease is due to avian or human type of bacillus". Microscopic examination was not done.

Comment.—The data on Case 2 are so sketchy that little can be drawn from them. Note that he still had a negative response to human old tuberculin in November, 1934, but a 3 plus reaction to avian tuberculin in January, 1935; two months later he began to run fever and death took place six months after discovery of the positive reaction to avian tuberculin. The questions arise—"Did he contract tuberculosis in addition to the congenital cystic condition? Was this tuberculosis responsible for the pyrexia and for the positive response to avian tuberculin?" It is impossible to say. Possibly, as with Case 4, he developed tuberculosis during his stay in an open ward. Feldman[4] quotes instances in which positive response to avian tuberculin is found in human type disease, so it would seem that the positive response in this case is in no wise diagnostic of the type.

The early death of Case 2 compared to the others may be significant of some added factor in his illness, but his last chest film shows similar shadows with similar distribution to what is seen in films of Cases 3 and 4, and this close similarity of film appearance is strong evidence against pulmonary tuberculosis being the dominant factor in his death.

Case 3

Male, aged 19 years. This boy was a sturdy infant but had had dysentery at two and was never strong after. He had his first clinic examination at the age of 7 when it was reported that, "he has no complaints but is small, pale and thin, with enlarged tonsils and tonsillar glands; his film shows no evidence of tuberculosis". He had annual examinations at the clinic until admission to sanatorium at 16. At 8 he had measles and his third film taken the next year was read as "generalized dirtiness with left root enlarged"; each succeeding film showed slightly more "dirtiness" until at the age of 12 he was diagnosed "moderately advanced tuberculosis". Tonsillectomy at 9 was followed by subsidence of the nodes in his neck, but his general condition continued much below par, he was "thin and pale" and at 12 weighed only 61 pounds; he had infrequent colds and with one cold only was any sputum reported: in 1940, and again in 1941, tuberculin tests were negative, but the chest shadows increased and he was finally advised to enter sanatorium. Admitted October 29, 1942 he continued ambulant until 4 days before his death on April 13, 1945. He died quietly complaining only of distressing dyspnœa and weakness. During his years in sanatorium he had no significant episodes; there was some "tightness" in his chest not associated with the dyspnœa. His only cough was a constant clearing of the throat and he had no sputum except a little mucus that was cleared from his vocal cords.

The main autopsy findings were as follows: Marked clubbing of fingers and toes. Pleural sacs; no effusion and no adhesions. Both lungs show the same condition; upper half to two-thirds of each lung is greyish white, surface uneven, nodular, firm with numerous small emphysematous bullæ. Lower third of each lung congested and dark red with most of lung tissue looking normal but small patches are affected. The lesions in both lungs are similar in kind and extent. The right lung has only two lobes; the left has its normal two lobes. The upper one-third of each lung contains no air, being solid, rubbery in consistence, greyish-white in colour with a few small cystic areas and very little caseation. The middle thirds show some areas of apparently normal tissue, but the greater part is like the upper thirds. The lower thirds show only scattered involvement.

Bronchi, normal. Blood vessels normal. Several tracheobronchial nodes appear as dark soft masses: these are not enlarged. No cardiac lesions.

Comment.—This patient is remarkable chiefly because of his uneventful course. He had the very definite illness at two but that is about all, except for malnutrition and asthenia associated with his polycystic disease. He mentions repeated colds at his various clinic examinations, but "dry cough" was the only constant symptom of these colds. He could recall only two occasions on which considerable yellow sputum was present for a few days. "Colds" came on after playing hockey or other games at school and it seems probable that these so-called "colds" actually were the sign of cardiac embarrassment caused by extra exertion; he was exceedingly flat-chested so that his vital capacity even in childhood must have been severely re-

stricted. This boy evinced keen intellect; the lassitude that characterized him physically was not evident in his mental acuity and scope.

No microscopic report is given; through error only a minute fragment of one lung was examined, and although the pathologist found indications of cystic disease he felt that he was not justified in making a conclusive report on so meagre a specimen; later, when he had had the material from Case 4 to work on he had no hesitation in making the diagnosis of congenital cystic disease.

Case 4

Male, aged 18. He was a healthy child with no illness of note prior to his first examination at the clinic August 31, 1934. His clinic record states: "Weight 35 pounds; no complaints; tonsils and posterior cervical glands moderately enlarged; lung markings prominent throughout but not suggestive of tuberculosis". He had measles that fall and at the clinic, September 1935, was reported to have had frequent colds throughout the year; his tonsils were now large and septic with increased swelling of the cervical glands; his film shows heavy roots with generalized dirtiness throughout both lungs, but most marked in left apex; he was called a "tuberculosis suspect". A year still later he had had his tonsils removed under ether but his cervical glands were still enlarged, he was having more frequent colds with cough and sputum and he "wheezed in his sleep"; the shadows in both apices were increased.

The development of asthmatic attacks went on apace and at the 1937 clinic his report was: "Age 8; weight 49 pounds; has not been well all this last year, but has had frequent asthmatic spells with temperature to 100°; coughing in the attacks that often ends with vomiting; he is very active and goes to school between spells; he is thin and pale and there are scattered wheezes throughout his chest; his film shows increase of shadows that now are somewhat miliary in appearance." He was classed as "moderately advanced tuberculosis."

He went into sanatorium for further examination and there his reactions to human and avian tuberculins were negative. He was sent home and attended school for a year and a half. He was readmitted July 29, 1939 to July 2, 1941. He was free from asthmatic spells for these two years but made no general improvement. Tuberculin response was negative on admission, but he was kept in a ward with open cases and at discharge gave a three plus response to 1/1,000 of human type old tuberculin. The next sixteen months he spent at home attending school, but had frequent spells of asthma that put him in hospital and when a film of September 30, 1942 showed double spontaneous pneumothorax he was sent into sanatorium October 29, 1942 to April 14, 1945; the spontaneous pneumothoraces were kept up artificially for two years but resulted in no appreciable benefit.

He left the sanatorium in April 1945 and visited in Winnipeg for two months until an asthmatic "spell" sent him into hospital for one month; he was said to be *in extremis* in this attack but it quickly subsided under morphine and the film taken then shows no sign of spontaneous or other change. He went home in August 1945 and continued ambulant for the following year. He grew worse but was free from severe asthmatic spells until taken with a typical one on September 22, 1945; he died the following morning.

Autopsy report.—The lungs were found densely adherent to the chest wall, except for the lower lobes anteriorly.

The right lung is voluminous, weighing 780 gm. in fixed state. Lung has two lobes only. Upper lobe shows diffuse spaces which for the most part are smooth lined, varying in size from 0.3 to 2.5 cm. The lower lobe shows similar changes, less marked, together with numerous emphysematous bullæ chiefly located along the free margins. There is some bronchial dilatation, most marked in the upper lobe. The left lung weighs 780 gm. Numerous enlarged tracheal lymph nodes are present, measuring up to 3 cm. in diameter. Some of these contain yellow caseous foci. The unsupported margins of the lung show marked emphysema. Scattered throughout the upper lobe are numerous smooth lined cysts, measuring up to 4 cm. in diameter.

The bronchi show moderate dilatation. The lower lobe is dark in colour, partially consolidated and shows bronchiectasis with a few cyst-like spaces about the periphery.

Microscopic.—Both lungs show the same changes. The numerous cystic spaces for the most part have no epithelial lining due to improper fixation; in areas, however, small amounts of flattened and cuboidal epithelium remain. Intervening lung tissues show rather marked fibrosis; some areas are completely fibrosed, in other areas the alveolar walls are greatly thickened. Areas of lung show fairly marked emphysema. Some alveoli and bronchi contain giant cells, but no evidence of tuberculosis is seen in multiple sections of the lung. Both lungs show fairly marked congestion with intra-alveolar hæmorrhage and numerous heart failure cells. Bronchial lymph nodes show areas of caseation surrounded by a zone of epithelial reaction (see Fig. 1). The presence of tuberculosis lymphadenitis of the bronchial nodes in this case probably represents a concurrent tuberculosis infection, but no evidence of pulmonary infection can be demonstrated. Cause of death in this case was right-sided heart failure due to congenital cystic disease of the lungs.

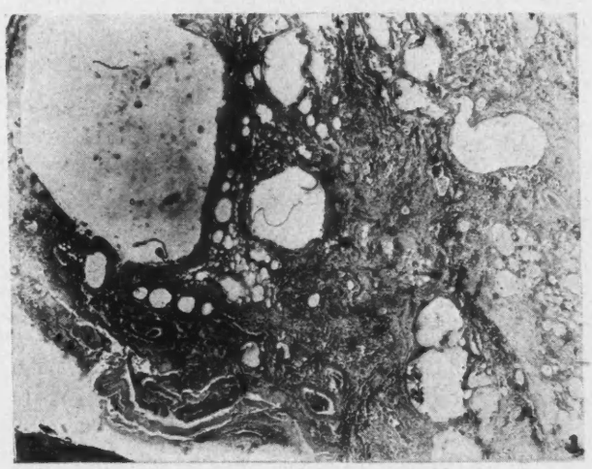


Fig. 1.—Low power photomicrograph (x 50) showing the cysts of the lung. Cysts have thin fibrous walls lined in part by a single layer of flattened and cuboidal epithelium. The intervening tissue shows marked fibrosis with some round cell infiltration.

Comment. — The concurrent bilateral spontaneous pneumothoraces in 1942 require explanation: why did he not have total collapse of both lungs and die? The explanation seems to be that he had had previous spontaneous collapses that resulted in the formation of adhesions in the basal areas sufficient to prevent collapse of those portions when both lungs collapsed at once. There is evidence that pneumothorax was the cause of adhesions because Case 2, who had had left-sided pneumothorax, had

adhesions on the left; Case 4, who had had bilateral pneumothorax, had dense bilateral adhesions; while Case 3, who had had no pneumothorax at any time, had no sign of adhesions in his chest. As judged by films and autopsy observations Case 4 had far more extensive cystic development than the other cases: was this due to the asthma, or was the asthma a concomitant of the cysts? Allergy tests might have been helpful in determining this point, but in any case, it seems certain that cystic formation was aggravated by the severe "spells" of asthma.

Discussion of Cases

Congenital cystic disease of the lungs is a condition much better known by the pathologist than by the clinician. It was only rarely that it was diagnosed outside the autopsy room and the pathological laboratory until radiography, and more particularly broncho-radiography, came to the aid of the clinician. The x-ray, a better appreciation of laboratory findings and shrewder evaluation of signs and symptoms makes it possible to improve differential criteria to the point where cystic disease may be diagnosed with a degree of assurance. Our record in the four cases dealt with here is far from brilliant, but we can offer in extenuation the fact that never before had familial incidence been encountered, so that an infection rather than a congenital condition was looked for.

A review of the literature indicates that our cases are unique in their familial incidence, in the presence of marked clubbing of fingers and toes without pulmonary sepsis, and in the malignant nature of the disease which resulted in the death of all four before adult life was reached; in all other characteristics they conform to the general picture of the alveolar type of congenital cystic disease.

Age of onset.—The condition was far advanced in Case 1 and in Case 2 at their first clinic examinations at the ages of 18 and 10 respectively; the first definite changes in the x-rays of the lungs were noted in Case 3 at 9 and in Case 4 at 6. It is thus seen that, with the possible exception of Case 1 the age of onset falls between 5 and 10. Schenck,[1] in outlining the embryology of the lungs, states that the process of division of cells that ultimately form the respiratory tree begins at the end of the fourth week of intra-uterine life and is not normally completed until about the eighth year of post-natal life. Pathologists seem to be pretty well in agreement that congenital cystic disease has its origin in some maldevelopment of the respiratory mechanism of the lung; the fact that our cases began to manifest disease about the time that the process of budding normally comes to an end is interesting and may be not without significance.

Antecedent disease.—Many investigators who accept the theory of congenital defect in the lung as the basic cause of cystic disease consider respiratory infections to be potent contributory factors. In this regard they give first place to the respiratory complications of measles. Three of our four cases, 2, 3 and 4 had measles *after* the first appearance of abnormal shadows within their lungs, so that measles appears to be ruled out as a factor in initiating cystic disease. The only antecedent respiratory infection that was reported in all four cases was the common "cold". We think what passed for "frequent colds" were but episodes in the symptom complex of cystic disease, precipitated by over-exertion, for when these boys were in sanatorium on supervised routine they did not have colds, nor did Case 4 have asthmatic attacks. A condition that was noted early in Cases 2, 3 and 4 was enlarged tonsils with enlarged nodes in the neck: each of the three had tonsillectomy shortly after having had measles and the enlargement of the nodes disappeared following tonsillectomy. It is not clear that this toxic condition of the tonsils and cervical nodes had any influence on the cystic disease, but it is a point for the record.

Cyanosis and pallor.—These conditions are discussed together because the ashy grey pallor noted in all four cases seems to have been an expression of the underlying cyanosis. That it was not due to anæmia or hæmoglobinæmia is evidenced by blood counts that show red cells over 5,000,000 and hæmoglobin content of 100% or more in 3 and 4. Case 2 had 4,800,000 red cells with 85% hæmoglobin while we have no record of No.1's blood findings. Cyanosis was present in all cases and became more marked as the cystic condition progressed. Extreme cyanosis was present in the terminal stages of all four. Cyanosis has been noted by practically all writers on cystic disease: expansile cysts enlarge until they displace the trachea or cause pressure enough to interfere with aeration; spontaneous pneumothorax can cause it; it appears in those cases in which asthma

is a characteristic, and in septic bronchiectasis types. All these produce recurrent and transient cyanosis but in our cases the condition was continuous and progressive. A closer analogy to the type of cyanosis observed in our cases is found in Ayerza's disease.[5] In respect to cyanosis, dyspnœa, finger clubbing and blood findings, Ayerza's disease and the alveolar type of cystic disease are strikingly alike. The underlying cause of these symptoms in Ayerza's disease is arteriosclerosis of the pulmonary arterial system with marked thickening of the intimal coat, thus cutting down blood volume and interfering with aeration of what little does get through. It seems that in our cases a similar cutting down of pulmonary circulation and non-aeration of blood occurred due to marked fibrosis surrounding pulmonary blood vessels and air sacs. Calma,[6] reports a case of cystic disease in which obliterative endarteritis was present.

Dyspnœa on exertion was a complaint of all our cases, but they were not aware of any distress when at rest. Case 4 had it severely with asthmatic spells and dyspnœa with marked cyanosis characterized all final episodes. In a general way dyspnœa kept pace with the increase of fibrosis noted in their chest films and, apart from Case 4's asthma, it apparently resulted from the same difficulties of aeration that caused the cyanosis.

Cough and sputum.—All cases reported having had frequent colds with cough and sputum, but observation did not bear this out. Only very occasionally was a mucopurulent specimen obtained and these were got during epidemic colds; in all cases resort had to be made to gastric lavage in search of material for examination.

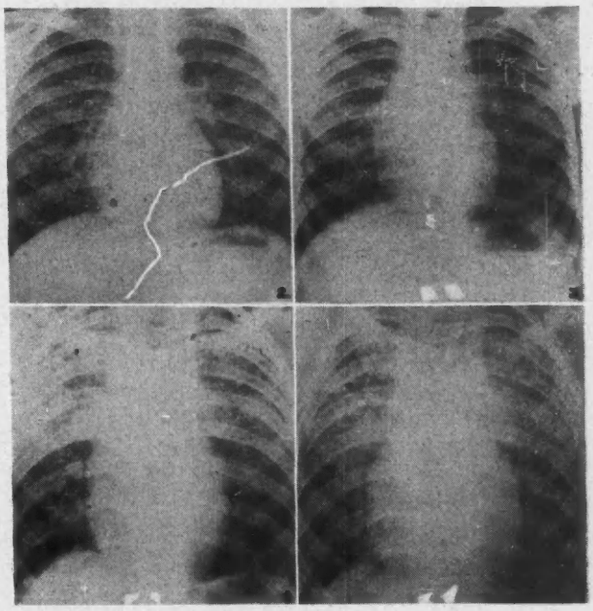
Pyrexia.—Case 2 ran fever of 100 to 101° for the last four months and Case 4 had fever of 100° with asthmatic attacks; apart from these all cases were without elevation of temperature.

Pulse rate and blood pressure.—There was no high pulse rate corresponding to the cyanosis and dyspnœa produced by difficult aeration and embarrassed cardiac action. There was quick and marked response of pulse rate to exertion, but it dropped back to its customary rate in approximately normal time. Case 1 was found with a pressure of 150/100 on admission to sanatorium. Cases 3 and 4 did have repeated tests made and pressures were normal for the average person on sanatorium routine.

Râles and wheezes.—Fine râles in the hilar areas were characteristic of all cases when first found with the disease: but in Cases 3 and 4 râles disappeared in later years. Case 3 had a silent chest in which nothing whatever could be heard; Case 4 had clicks and expiratory wheezes scattered all over his chest and these findings conform with the autopsy findings in each.

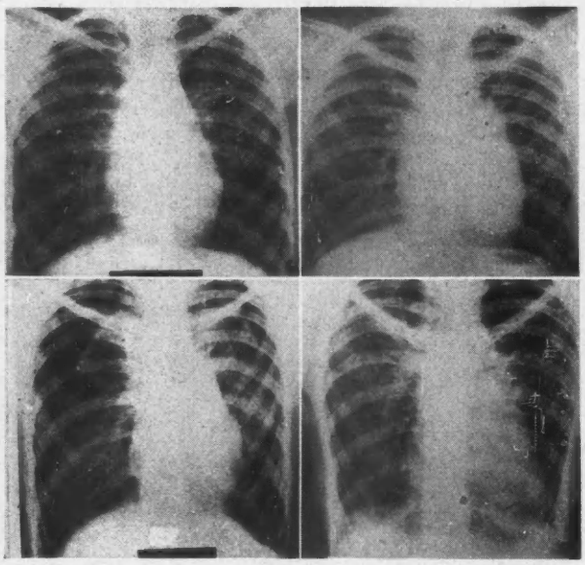
Blood counts. — Competent studies of blood were not made. White cell counts that were done gave figures of 10,000, 13,000 and 11,000 in Cases 2, 3 and 4 respectively; in Cases 3 and 4 the lymphocyte percentage was 60 and 76.5 respectively, but these phenomenal findings were were not verified.

Radiograms.—The accompanying illustrations are selected from a series of films of Cases 3 and 4. They illustrate the gradual and steady progress of the disease. In all cases the apices of the lungs appear to be the worst with a gradual diminution of disease towards the bases, and this appearance on the films is borne out by autopsy findings. The upper left lung is usually mentioned in film readings as showing more disease than the right; this is borne out in the pathologist's report on Case 4. It is not clear why there should be a preponderance on the left, nor is it clear why the upper part of the lungs should bear the brunt of the disease.



Figs. 2, 3, 4, 5 illustrate progress of disease in Case 3. Fig. 2 was taken in 1933, Fig. 3 in 1939, Fig. 4 in 1943 and Fig. 5 in 1945, only two days before death and shows the final degree of cardiac engorgement.

Diagnosis. — The recognition of congenital cystic disease of the lungs is not always likely to be so clear-cut as the syndrome pictured here because the basic features of the disease are often masked by complications. The bronchial type is frequently over-laid by pulmonary sepsis; asthma may dominate the picture so completely that nothing else is manifest; an infected solitary cyst may present itself as a lung



Figs. 6, 7, 8, 9 illustrate progress of disease in Case 4; they were taken in 1938, 1940, 1942 and 1946 respectively. Fig. 8 shows the double spontaneous pneumothorax at time of detection before any refills were given. Fig. 9 shows the lungs after re-expansion and was taken seven months before death.

abscess; and concurrent pulmonary tuberculosis will most effectively screen all basic characteristics of other disease. We failed to make a clinical diagnosis in our four cases, although there was no masking of the basic characteristics; it may be that we were thrown off the track by the familial incidence, but one doubts if we would have done any better if they had presented themselves to us singly. The fault obviously lies in the fact that we have been content to let the pathologist make the diagnosis for us, or to let these cases go into the catch-alls of pulmonary tuberculosis and pulmonary sepsis. The clinician must enlarge the scope of his understanding of diseases of the chest and question every diagnosis of tuberculosis that is not confirmed by microscopic section or bacillary findings.

Grateful acknowledgment is made of hospital records on Cases 1 and 2 made available by Dr. A. C. Sinclair of St. Boniface Sanatorium.

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EARLY POSTOPERATIVE WOUND DISRUPTION

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DISRUPTION of the abdominal wound is a complication as capable of causing concern as any. Statistics reveal that the incidence of this condition is unexpectedly large, and is quoted by authorities as varying between 1 and 3% of laparotomies.[1, 2] Of more serious importance is the high rate of mortality associated with it. This is said to average about 33%. A study of the recent records of the Winnipeg General Hospital reveals that of 18 cases requiring resuture of disrupted wounds there were 6 deaths which can be attributed directly or indirectly to this catastrophe. The majority of these cases represented major surgical problems, and the procedure was performed on patients in whom it was impossible to obtain an optimum surgical state. The age of the patients varied from that of an infant three days old to adults who were advanced in life. The time of occurrence of the incident was as early as 3 days, and as late as 39 days after the operation. Post mortem examination was obtained in 5 instances. It is interesting to note that peritonitis was present in only one case (Table I).

TABLE I.

WOUND DISRUPTION AT THE WINNIPEG GENERAL HOSPITAL

Number of cases of secondary suture	18
Site of incision	
(*a*) Upper abdomen	13
(*b*) Mid-abdomen (Transv. 2)	3
(*c*) Lower abdomen	2
Days post-op.	3-39
Mortality	6 (33 1/3%)
Cause:	
(*a*) Pulmonary embolus	2
(*b*) Myocardial infarct	2
(*c*) Peritonitis	1
(*d*) Uncertain	1

REASONS

The reasons for this disaster have been capably examined and are well known. They include poor suture technique and, to a less extent, poor suture material. It has been shown that wound disruption occurs with all types of suture material. Failure to obtain proper abdominal wall relaxation for the facile closing of the wound was once a problem frequently encountered, but now should rarely be seen with the fine advances in anæsthesiology. The occurrence of increased intra-abdominal pressure due to coughing, vomiting and distension is better controlled by new methods of preoperative preparation of chronic pulmonary states and the postoperative use of the indwelling gastric suction which is now perhaps universal. Surgery in the obese, when elective, is recognized as safer when the weight is reduced, thus providing among other advantages a lessening in intra-abdominal tension. There can be no doubt, since most wound disruptions occur in the upper abdomen, that the strong, lateral pull produced by the costal margins because of abnormal respiration and coughing is a factor (Table I).

DELAYED HEALING

These and other reasons no doubt all contribute to the development of wound dehiscence. However, it is most reasonable to accept the opinion of certain investigators that some element of a general nature is present in the tissues which influences wound healing. The problem of failure of wound healing may actually be considered a subject of its own. It has been stated by Colp,[3] that "wound dehiscence will never be an unavoidable surgical complication until methods are available to differentiate accurately those patients with poor reparative powers and until means are at hand which will unfailingly promote the prompt healing of tissues in these specific cases." This statement was made in 1934, but the principles involved in proper wound healing were noted as early as 1919 by Clark.[4] His studies revealed that the repair of wounds in dogs began at once provided the animals were fed on a diet rich in proteins. A most excellent piece of investigation on the problem of failure of wounds to heal was produced in 1938 by Ravdin and his associates.[5] Sections of wound edges in dogs in which a protein deficiency was induced demonstrated abnormal œdema, failure of fibroblast proliferation, and lack of vascular ingrowths, in contrast to animals normally fed where wounds showed usual œdema, much cellular and vascular activity and the fibroplasia was normal. The wounds of the control dogs healed well and in the proper time, in contrast to delayed separation, and often evisceration among the dogs where the level of the protein values was reduced to a critical state.

Malnutrition is encountered frequently in cases requiring surgery in the presence of lesions of the biliary and gastro-intestinal tracts where vomiting and obstruction prevent proper intake of food. Similarly, protein deficiency results from diarrhœa associated with ileitis or ulcerative colitis where absorption of food is reduced. Yet it is interesting to learn that wound disruption is rarely encountered among the natives of the lower classes in the Principality of Madras, where it is stated that the protein portion of the diet is at a particularly low level.[6] The problems of nutrition and wound healing, therefore, would appear to be related, and probably protein deficiency is sufficiently important to justify a proper program of treatment to correct it when possible prior to undertaking major surgical procedure.

The earliest evidence of wound disruption is usually the appearance of a thin bloody fluid which may be sufficient to soak or stain the dressing of a hitherto non-draining wound. Pain is usually of little consequence. If this warning sign is disregarded, it is likely the wound will go on to an extensive disruption. The warning may not appear significant, as in the early phase of separation the skin edges may be in apposition except for a small opening. It must be remembered that in disruption it is the deep layers which first separate, and only in the final phase are the skin edges apart. If early recognized, the extent of the damage may be limited by the firm application of adhesive tape and measures taken to reduce coughing or distension.

TREATMENT

If frank disruption has occurred, a critical condition is present which demands immediate attention. Frequently this occurs in patients who have been ill for a long time prior to operation or have endured a stormy postoperative course, with the result that this further experi-

ence may shock them. They are apprehensive and perhaps in pain. Morphine should be soon administered in an appropriate dosage. The circulation should not be permitted to fail and intravenous fluids should be given. Modern experience reveals that blood and plasma are of unlimited value in such depressed states. The further treatment of these cases depends upon certain factors.

There is usually the surgeon's desire to close the wound quickly in order to prevent infection, herniation and a long convalescence. Certain individuals will tolerate this emergency procedure very well, but it must be kept in mind that in the face of shock and malnutrition secondary closure is a formidable proposition. No case should be treated without preliminary sedation and sufficient time to overcome or forestall shock. Very little should be done to the wound while the patient is in the ward. When all is ready, the patient should be transported to the operating room in his bed to reduce manipulation and draughts.

In the operating room a light general anæsthetic may be employed, and intravenous pentothal sodium is satisfactory. Later it may be supplemented with local anæsthesia. With the patient asleep or semi-conscious, the dressings are removed. Under aseptic precautions any loops of bowel which are exteriorized are separated from the wound edges and replaced in the abdomen. The skin field is now sterilized and the drapes applied. Novocaine 1% is infiltrated under the skin edges, and then the needle is directed into the pre-peritoneal layer and further novocaine is administered. Sufficient relaxation is generally obtained with this combination. Spinal anæsthesia presents the disadvantage of perhaps a drop in blood pressure in an already critically ill patient, but provides the best relaxation.

As to the actual closure, many methods have been described. The following is a method found to be satisfactory: The skin is separated back from the wound edge for a distance of one and a half inches. The further dissection of the anatomical layers which have become fused together from the primary operation is inadvisable. Too much time is consumed. The individual layers are too friable to be sutured to each other and are further injured by such treatment.

Figure of eight sutures are employed, using a double strand of silk-worm gut. Actually the superficial loop is missing as the ends of the sutures are tied parallel to the side of the wound over a metal Wetherbee plate. The deep loop of the suture embraces the full thickness of the abdominal wall apart from the skin. The suture is started through skin about 1½ to 2″ from the wound edge on a large cutting needle. It is then carried across the wound to the opposite side, down through the anterior aponeurosis, muscle and peritoneum. The suture is then returned to the original side and brought upwards through peritoneum, muscle and fascia. Finally it is carried across the wound again and out through the fat and skin on the opposite side. The sutures, having been placed about one inch apart, are now tied to the corresponding ends of the neighbouring suture over the Wetherbee plate. In this way the sutures do not cut through the skin. While the deep layers are compactly closed, the skin edges are left moderately separated, and free drainage is permitted. By this procedure the deep layers are opposed without leaving absorbable suture material in the depths of the wound.

ALTERNATIVE METHOD

All cases should not undergo resuturing. There are cases which die following secondary closure which I am sure would have survived if a more conservative procedure had been followed. I know of two cases in the past year who died of a complication apart from their original disease and the ensuing operation. Both did well for 6 or 7 days following their abdominal operations, one for repair of an œsophageal hiatus hernia and the other a subtotal gastric resection for ulcer. Both were over 60 years of age. Each patient developed an extensive myocardial infarction shortly after his secondary closure and died. There can be little argument that the suturing procedure subjects an already sick patient to an operation of considerable magnitude. To some of these cases the disadvantages of suturing outweigh those of some less extreme method of restoring wound closure, even though the greater possibility of hernia formation or extended convalescence is considered.

An alternative method of management of wound disruption is worth considering. Therefore, in certain cases where an emergency

operation might obviously spell disaster the coaptation of the skin edges by adhesive tape is a worthwhile measure. The procedure should be done under sterile precautions, and after any escaped loops of bowel have been replaced in the abdomen the wound is covered by a light gauze dressing. Strips of wide adhesive tape are applied in an over-lapping manner from the lower end of the wound upward, the ends of the tape being applied well around to the sides of the abdomen. Later when there is need to change the dressing, the tape may be cut away down the centre leaving the remainder as piers on which fresh adhesive can be placed to hold the new dressing in place. Tension can be increased by an assistant holding the sides of the abdomen while the tape is applied.

Occasionally when the wound edges are brought together in this manner healing may occur as satisfactorily as when the wound is sutured. There is little doubt that coaptation in most cases will be slow and incisional hernia will be more certain. However, the final effect may be a living patient in contrast with the possible fatal result of a more heroic procedure.

The further care of the patient with a disrupted wound, no matter what the closure, should be as strict or more so than for the first operation. Penicillin should be administered routinely for at least 48 hours. Decompression of the stomach should be maintained by use of a nasal catheter until danger of obstruction by mechanical or infectious means is over. States of anæmia, avitaminosis and protein deficiency must be avoided or treated vigorously. Blood transfusions, plasma and parenteral amino acids may be employed to maintain the patient's nutrition and improve his healing powers, and perhaps shorten his convalescence. Large doses of vitamin C, which is so useful in promoting wound healing, should be given.

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A COMPARATIVE STUDY OF ETHER AND SPINAL ANÆSTHESIA FOR SPLENECTOMY*

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THE object of this paper is to compare the operative and postoperative courses of patients undergoing splenectomy in so far as the anæsthetic is concerned (using ether and spinal analgesia). The material for this series of cases is from the records of operations of the main operating room at the Royal Victoria Hospital dating from 1931 to the present. Twenty-two cases were studied; 11 done under ether, the rest under spinal anæsthesia. The first splenectomy under spinal anæsthesia recorded here was done in 1936. Since then, it has been used in nearly all cases.

CHOICE OF ANÆSTHETIC

Pemberton[1] states the success of the operation may well depend on making the proper choice of anæsthesia. It must first of all be safe; also it must afford complete relaxation. Thirdly, it has to last long enough to insure completion of the operation. The surgeon may meet with difficult removal due to adhesions resulting from the perisplenitis in such a disease process as Banti's syndrome. Complete relaxation, therefore, is most important. He chooses nitrous-oxide, oxygen and ether administered by the endotracheal tube, or spinal anæsthesia.

Experimentally, ether causes contraction of the spleen[2] which is important in facilitating its removal and conserving the patient's blood supply (as a result of squeezing its contents into the circulating blood volume). Under spinal anæsthesia the spleen of the dog dilates 3 to 4 times its normal size.[3] At operation such a tendency has been counteracted by injecting 10 minims[4] of adrenalin into the splenic artery or pulp. This causes a noticeable contraction in the size of the spleen, gives the surgeon better access to the posterior gutter and retracts it from the diaphragm. It also serves as an autotransfusion of blood as mentioned above.

The conditions for which operation was undertaken were:

* From the Department of Anæsthesia, Royal Victoria Hospital, Montreal.

Hæmolytic jaundice	6 cases
Essential thrombocytopenic purpura	7 "
Banti's disease	6 "
Aplastic anæmia	1 "
Gaucher's disease	1 "
Exudative and proliferative splenitis	1 "

The average duration of operations in the ether series was about 50 minutes; of the second group, 70 minutes.

ANÆSTHETIC TECHNIQUES

In the ether series 7 cases were induced with nitrous oxide, oxygen and maintained by open drop ether. Four cases had open ether administered only.

In the second series 9 anæsthetics were done by the modified Etherington-Wilson technique[5] using nupercaine. One case was done under novocaine and one using pontocaine by the continuous method of administration.

following tonsillectomy in 1931. This was followed by a quinsy. A neutropenia was discovered suggesting panmyelotoxicosis. She died following splenectomy. No. 9 had a hæmoglobin of 21% on the operating day. All attempts to improve the blood picture failed.

The only patient who had a poor operative course was No. 6 who lost a considerable quantity of blood at operation. She was in a state of shock in the operating theatre and on return to the ward. No analeptics or transfusions were given.

Postoperative complications in which the anæsthetic may have played a part were cases 3, 6 and 8.

The age group is higher. All the patients in this group with the exception of Nos. 2 and 4 were in good preoperative condition. Number

TABLE I.

COMPARATIVE STUDY—ETHER SERIES

Year	*Case*	*Disease*	*Age*	*Supportive measures at and following operation*	*Convalescence*
1931	1	Thrombocytopenic purpura	5		Persistent vomiting 1 day. Low grade temperature 3 days.
1931	2	Thrombocytopenic purpura	6		Uneventful.
1931	3	Banti's disease	25	Intravenous saline at operation	Atelectasis diagnosed 4 days post-operatively. Resolved without treatment.
1931	4	Thrombocytopenic purpura	43		Low grade temperature—9 days.
1932	5	Thrombocytopenic purpura	12		Low grade temperature—28 days.
1932	6	Banti's disease	26	Blood loss at operation. Shocked condition on return to ward. No transfusion or analeptics.	Continuous temperature for 23 days. Consolidated left lung base suggestive of pneumonia.
1932	7	Banti's disease	13		Postoperative hæmorrhage into stomach. Continuous temperature —24 days.
1932	8	Exudative and proliferative splenitis	17		Developed lobar pneumonia, pericarditis with effusion, subphrenic abscess and purulent peritonitis —died.
1938	9	Panmyelotoxicosis (Aplastic anæmia)	16		Postoperative bronchopneumonia. Died 8 days later.
1939	10	Gaucher's disease	7	Postoperative blood transfusion	Some postoperative vomiting of blood.
1942	11	Banti's disease and bilateral cystic kidneys.	14	Postoperative blood transfusion—10th day	Postoperative temperature—12 days.

The age group is lower. Younger patients withstand this operation better.[6] There were only four cases in this series which were considered good operative risks. These were Nos. 3, 4, 6, and 10.

Cases 1, 2, 5, and 11 were considered a "B" grade risk. Cases 7, 8 and 9 were classed as poor risks. Case 7 was very anæmic. Her hæmoglobin was 20% three days preoperatively. There was no record of her condition on the day of operation. No. 8 had rheumatic fever 2 had had an ileostomy of two years' duration closed one month before operation. Her hæmoglobin was 77% at operation. She was considered a poor risk (Class "C"). The operative course was good.

No. 4 was described as an "emaciated white male, aged 70". On the date of operation the hæmoglobin was 48%. He was diagnosed as having pernicious anæmia, hæmolytic jaundice and carcinoma of the pyloric region of the stomach. He was classed a poor risk. The oper-

TABLE II.

SPINAL SERIES

Year	*Case*	*Disease*	*Age*	*Supportive measures at and following operation*	*Convalescence*
1936	1	Banti's disease	47	Postoperative transfusion 535 c.c. blood	Postoperative temperature, 7 days.
1938	2	Hæmolytic jaundice with cholelithiasis and amœbic dysentery.	25	Adrenalin minims 10 injected into spleen reduced it by about one-third volume. B.p. drop from 120/80 to 80/60. No analeptics used. B.p. 110/72 on return to the ward.	Vomiting intermittently for 2 days. Some recurrence later.
1939	3	Thrombocytopenic purpura	25	Adrenalin injected caused contraction of spleen. Transfused with 500 c.c. blood at operation, and with 1,140 c.c. of same postoperatively.	Good recovery.
1940	4	Hæmolytic jaundice and pernicious anæmia.	70	Adrenalin minims 10 into spleen caused marked shrinkage. Poor operative course. Glucose-saline 700 c.c. and plasma 200 c.c. were given I.V. with oxygen intratracheally. Vomited bile. B.p. preoperatively 116/64 considered high enough for spinal anæsthesia.	Died. Postoperative shock and bronchopneumonia.
1939	5	Hæmolytic jaundice	36	I.V. infusion of glucose-saline 750 c.c. Adrenalin minims 8 raised blood pressure and pulse.	Postoperative temperature of 101°. Diaphragmatic pleurisy.
1942	6	Hæmolytic jaundice	14		Wound infection.
1942	7	Banti's disease	25	Blood transfusion at operation 600 c.c.	Wound infection.
1943	8	Thrombocytopenic purpura	12	Operative transfusion of blood 300 c.c. and plasma 500 c.c.	Wound infection.
1944	9	Hæmolytic jaundice	9		Temperature postoperative for 5 days.
1946	10	Thrombocytopenic purpura	17	Operative blood transfusion 500 c.c.	Postoperative temperature 3 days.
1946	11	Hæmolytic jaundice	31	Operative blood transfusion 1,000 c.c. Adrenalin minims 10 into spleen reduced its volume.	Good recovery.

ative course went well in all cases with the exception of No. 4. The anæsthetic may have figured in the convalescence of Nos. 4 and 5.

CONCLUSIONS

The second series of operations, having been done in the last ten years had the advantage of recent developments in preoperative preparation and operative and postoperative care and support (which the first group did not have). This may account for the apparent better convalescence in the second group of cases.

Spinal anæsthesia provides the optimum, unvarying relaxation so necessary to aid the surgeon in difficult removal of the spleen. It is hard to equal by other techniques such as cyclopropane-curare, which requires greater skill of administration. Also, cyclopropane causes the spleen to dilate.[7] The use of adrenalin is contraindicated with this agent.[8]

The safety of spinal anæsthesia for the operation of splenectomy is demonstrated by this series of cases as well as by its almost routine use in this hospital for other major upper abdominal surgery, such as gastrectomy, common duct exploration, etc. It fulfills the third requisite of Pemberton, mentioned above, *i.e.*, sufficient duration, which may be assured by using the long-lasting drug nupercaine, or, pontocaine by the continuous technique.

SUMMARY

The operative and postoperative courses of 22 patients undergoing splenectomy were studied from an anæsthetic standpoint. Eleven cases were done under ether anæsthesia; the remainder with spinal. The results are comparable, with spinal anæsthesia affording the greater operative ease due to the marked muscular relaxation and contracted bowels in a quiet abdomen.

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REPAIR OF BONE LOSS AND FRACTURES OF THE FACE AND MANDIBLE WITH CANCELLOUS BONE CHIPS

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REPAIR of fractures of the bones of the face by means of cancellous chips has in the past few years become so popular that we tend to feel that it has long been the operation of choice. However, as far as I have been able to discover, Mr. Mowlem of the Middlesex Hospital, did the first experimental work with this method in 1941. Massive cancellous grafts had been used successfully prior to this time, and had shown in comparison to cortical grafts a markedly increased tolerance to infection and a more rapid structural adaptation. Mr. Mowlem argued that these two characteristics pointed to a more early vascularization of the cancellous transplant and to increased cellular activity of the bone cells. On this basis he felt that by increasing the surface area of the graft through fragmentation, a much larger number of bone cells would become accessible to an early blood supply and more rapid fusion would result.

In using the cortical graft it is considered necessary to wait three to six months after the last sign of infection, before grafting can be carried out. In the cancellous chip method, grafts have been done in the presence of infection.

If while inserting a cortical graft an opening is made into the mouth during the preparation of the graft bed, the operation is of necessity discontinued and put off for two to three months, as infection from the mouth would cause loss of the graft. With the cancellous chip method, grafts have frequently been successful where there are known openings into the mouth, and aside from taking certain precautions the operation proceeds according to plan.

With chips it is possible to build out contour much more easily and with much less experience than is necessary in the planning of a single cortical graft. Chips are used without wire fixation and therefore can be applied where massive grafts cannot be used, as in skull defects. However, chips do not support the bone ends in place as does the solid graft and some extraneous method must be used.

In all cases the source of bone is the same, although the method of obtaining it may vary according to the individual wants. An incision of the required length is made over the crest of the ilium just posterior to the anterior superior spine and extended down to bone. By means of dissection and the periosteal elevator the crest and inner and outer plates of the iliac bone are cleared with difficulty of their muscular attachments. Then using an osteotome, thin sheets of bone 2 to 4 mm. in thickness are cut from the crest, as though one were planing a board. This continues until sufficient cancellous sheets have been obtained. Originally, complete blocks were very carefully cut from between the tables to be later cut into chips and this method is still of value in certain cases, as it disturbs the muscle attachments very little and allows a more rapid return of the patient to normal activity. However, it is time-absorbing and is feasible only where small amounts of bone will suffice.

In the upper part of the face where repair is mainly restoration of contour for cosmetic reasons, the sites usually needing attention are: the bridge of the nose; supraorbital region; malar zygomatic region; frontal region; infraorbital region; cranial region.

The nasal repair is a long procedure and as a rule requires many steps. As this would need a paper in itself, the procedure will not be dealt with here. However, it is similar to contour repairs in that the final step is repair with a planned cancellous bar along the depressed bridge. Chips are not as a rule used here.

Elsewhere on the face and scalp the procedure is in the main similar in all areas. An incision of the required length is made and is deepened to the level at which the graft is to be inserted. The bone ends or edges are cleared and are usually freshened by means of a rasp or file. Occasionally a very small opening may be used and the deeper areas opened up by blunt dissection. In any event it is advisable to leave a graft bed which can be closed over the chips deep to the skin. The correct contour is obtained by building up chips to the required depth and if possible, slightly over-correcting. The chips in these cases are held in position by the tissues sutured over them. Pressure bandages are applied.

It is in repair of the non-united or partially lost mandible that cancellous chip grafts have

been most widely used during the past war, and the steps of procedure as adhered to by most surgeons are as follows:

Preparation of the area.—In a badly compound comminuted fracture of the mandible where there is such loss that it is realized a graft will be necessary, much time can be saved by surgical removal of all loose and small sections of bone, and extraction of all teeth in the area of fracture. Drainage should be established and therapeutic local and general steps taken to clear up infection as soon as possible. Any skin loss must be made good by local flaps or distant pedicles. Free grafts are not used in these cases.

As far as possible, remaining bone fragments are splintered so as to retain their normal position. This, if well done, will save much time and inconvenience at the later operation, and assures a much more satisfactory result. The malpositions to guard against are the forward inward, and upward riding posterior fragment, and the downward and backward pull on the symphyseal end. Metal cap splints on upper and lower maxilla are used where there are teeth with a saddle to the posterior fragment.

In edentulous patients a guttapercha gunning or gutter splint holds the upper and lower jaws in approximate position. Occasionally the posterior fragment may be best controlled by a head cap and bar or a cap splint and bar with a stainless steel wire through the angle of the jaw.

Each case is a problem in itself and requires considerable ingenuity to attack it.

At time of grafting, a more solid type of fixation must be used; as a rule where adequate teeth are present, this consists of cap splints to upper and lower jaws with locking devices. From the lower splint a bar is fixed so as to curve backwards beyond the angle of the mandible on the affected side. Clauston-Walker pins are then drilled into the posterior fragment and are fixed with a universal joint.

All locking devices on the splints are now removed by a dental or assisting surgeon. This is necessary as otherwise chiselling or nibbling the bone ends is almost certain to cause the cap splints to come off.

An incision is made along the lower border of the mandible in the region of the fracture, and the bone ends of the fragments found and dissected clear of surrounding tissue. Do not clear tissue further than is necessary or the blood supply will be impaired. Do not, if avoidable, open into the mouth. However, if this happens find the opening and carefully suture it.

The bone ends are now chiselled and nibbled back past the compact eburnated surface to bleeding cancellous bone and a shelf is established on the outer surface for the chips to "take" on. At the same time a pocket is made behind each bone end to engage a supporting plate, of cancellous bone.

At this stage the assisting dental surgeon goes into the mouth under the drapes, re-locks the locking plates, and by means of backward pressure on the angle establishes the normal relation of the two ends. The universal joint already on the pins is now fitted over the bar running from the lower cap splint, and the joint screws are tightened in this position.

The bone is now taken from the iliac crest, and using osteotomes, is cut into one or more plates, and many chips. The plate is 1 cm. by 2 mm. thick and long enough to bridge the gap and overlap behind each fragment by 1 cm. The chips are roughly 2 mm. x ½ cm. x 1 cm. The hip wound is closed and dressed. Routinely I use a drain which I leave in for 48 hours.

The bone plate is fitted firmly behind the bone ends but is not tied or wired into position. Chips are then placed in irregular fashion along the plate surface extending well on to the shelved surface at both ends. Sufficient are used to build the area out to normal mandibular contour. Note that chips are irregularly placed, not in brick-like fashion. Space for hæmorrhage between the chips is encouraged, as only thus do we increase the graft surface area. Use sufficient chips to over-correct the defect. The deep tissues are now sutured over the chips to hold them in position and the surface wound is closed. A firm bandage is fixed about the mandible keeping firm, even pressure over the area.

There are certain points to be noted throughout this procedure. The taking of the bone from the iliac crest is a separate operation. It is not done prior to preparation of the bed and fixation of the bone ends because until the bone ends are fixed, the length of plate needed is not known. However, with the splint in position, the bone gap is measured by caliper and 2 cm. added for overlap. Now the surgeons and nurse

re-scrub and re-gown, use a complete new set of instruments, and take the bone graft. Infection of the hip is not uncommon where suitable precautions are not taken, and when it occurs it is a difficult condition to clear up. Following operation, the patient is kept quiet in bed for 10 days to prevent hæmatoma formation.

Before suturing the skin wound, if troublesome oozing is occurring, it is wise to drain the mandibular wound. With careful dressing, sepsis should not occur, whereas a large hæmatoma in the graft area can be very troublesome. If you have not drained and a large hæmatoma does occur, evacuate early before it can become infected.

Where infection is expected—as when the mouth has been entered—leave two small tubes in the mandibular wound and inject 4,000 units of penicillin in 2 c.c., twice a day, for five days.

Routinely the stitches are removed in 6 to 8 days, and pressure dressing comes off at this time. At four weeks, all locking bars are taken off the splints and the graft is tested carefully for union. If this is not firm, the jaws are re-fixed in position. Tests are made every two weeks until union is firm.

Clauston-Walker splints are left on from four to eight weeks, depending on need of fixation and infection around the pins. If the pins must be removed early in the fracture at the angle type, a saddle may be fixed from the lower mandibular splint back to the anterior border of the ascending ramus.

Shortly after Mr. Mowlem's original paper on the subject came out in 1944, we had a group of ten patients needing mandibular graft and it may be of interest to describe the troubles we had following a new method, and what results we achieved with it. There was a certain amount of difficulty present during the treatment of these fractures, not normally encountered. The main part of our unit and patients was situated in the plains of India during the hot season, but I had managed to secure thirty beds in a hill station three hundred miles away where we could operate on our major surgical cases. Patients were moved up and down for operation and during convalescence. I operated in the hills for ten days and returned to work in the plains for ten days, during which time the postoperative care was undertaken by various medical officers, some of whom were not adequately trained in observing results and in instituting necessary treatment. Our dental laboratory was in the plains, so that splints had to be made and be cemented on there, with no hope of last minute re-adjustment. These factors affected our results.

CASE 1

Non-union in body of mandible with no loss of bone.

May 13, 1945—Operation. Fracture was found with difficulty. Short plate and three small chips were inserted. Single cap splint with bar was used.

May 23—Returned to plains, leaving patient in hills.

June 3—Returned to hills, and found posterior splint has been off ten days. Re-cemented splint.

July 6—(Two months.) No clinical or x-ray union.

August 7—No union. Lower splint retained but intermaxillary fixation discarded for first time.

September 20—No bony union but fibrous union appeared satisfactory, and patient was discharged.

In this operation there was not sufficient removal of compact bone and there was no immobilization for ten to fourteen days during the most necessary period.

Dental co-operation and skill is important, as fixation must be sure. Remember the graft gives no support to the bone ends for the first three weeks.

CASE 2

This was a fracture of the body of the mandible. Clauston-Walker splints were used and a 1¾″ gap was filled by 12 chips. Drainage was established.

At completion of operation, it was found that there was an unclosed opening into the mouth. Penicillin 4,000 units twice daily, were given for five days. Discharge was copious from both internal and external wounds, and two months after operation three necrotic chips were removed at open operation. The remaining chips were firmly united in a viable bridge of bone.

The discharge cleared a few days after this operation, and when last seen, three months after the original grafting, union was occurring.

CASE 3

A bone graft of body of mandible done six weeks after infection had cleared. Penicillin was used for five days. There was springy (*i.e.* clinical union) after four weeks.

CASE 4

Mouth was entered at initial operation, so operation was deferred for one month. At operation, the posterior fragment was freed with difficulty. A 2″ plate and 15 chips were used. Penicillin was injected locally for 5 days. The pins were loose and out in four weeks. There was firm union in eight weeks.

CASE 5

Bone graft to symphysis region. Closed without drainage. Saddle and Clauston-Walker pins removed in four weeks. Firm union in eight weeks.

CASE 6

A symphysis fracture closed without drainage. Springy union in four weeks. Pins retained for six weeks.

CASE 7

Cap splint and bar only used; 1¼″ plate and 14 chips. No drainage. Clinical union in four weeks.

CASE 8

A bone graft back nearly to the angle. This fragment had ridden forward, inwards, and upwards so far that it was impossible to bring it back into its proper position without very free dissection of all surrounding tissues. A 2½" plate was used and 12 chips inserted. The area was drained.

Because of the wide dissection we were afraid the posterior pins might connect with the graft area and possibly infect it; however, there was no infection, and springy union was present at the end of four weeks, at which time the pins were removed. Two months later there was still not solid union, probably due to the upset in local blood supply.

CASE 9

This case was attacked late and although clinically there was no union at all, the fibrous tissue was so thick that it was with great difficulty that we found the line of fracture. However, when the compact bone ends had been cleared away there was a 1½" gap which we closed with a 2½" plate and twenty (20) chips.

The main part of the unit returned to the plains and eight days after operation, the patient developed a tense swelling over the mandible, showed increased fever and pulse rate, and soon started a copious discharge from a sinus into the mouth. This was probably the result of minimal infection introduced into the graft area as mouth openings usually showed up much earlier. Penicillin was injected once locally at this stage and the wound was opened. No pus was found.

Four weeks after the initial operation, three chips worked out of external sinus. X-ray showed three more chips sequestra but the other fourteen chips appeared viable. At this time we injected 5,000 units of penicillin twice a day for five days. Three weeks after the three chips discharged, the sinus healed and there was no further discharge. Three months after operation there appears to be x-ray union.

CASE 10

Uneventful union in eight weeks.

SUMMARY

1. Only one known failure: In this case there had not been sufficient cutting back to normal bone and fixation had not been adequate due to splint coming off.

2. In three cases there had been an opening into the mouth. In two cases the opening was not closed and massive infection occurred. However, with local penicillin injection the infection cleared up. A few chips were discharged and the cases went on to a slow union.

In one case the opening was closed and the operation was discontinued for one month. One month later the operation was carried out with no bad effects.

3. Five cases were not drained, *i.e.*, there was no fear of hæmatoma formation. Four of these cases went on to uneventful union. In one the mouth had apparently been opened into, and there was sinus formation. On the whole, we felt happier about those cases in which we did not have to use drainage.

4. Results: (*a*) one failure; (*b*) one slow or not proved union, infected and discharging; (*c*) one had union in 3 months, infected and discharging; (*d*) three had clinical union in four weeks; (*e*) two had clinical union in five weeks; (*f*) one had firm union in 7 weeks; (*g*) one had firm union in 8 weeks.

WORKMEN'S COMPENSATION*

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ALTHOUGH there are some differences between the Provincial Acts, the principles of workmen's compensation in all the Provinces are the same, and the problems of administration in each Province are remarkably similar. Our funds are what are termed as state funds, and come from the employers as assessment on payrolls, and trust funds from this source are set aside and invested to provide for future payments to men who are permanently disabled and to dependents of men killed in industry.

It is our task to receive the casualties of industry, supply them with the necessary medical attention, and endeavour to rehabilitate those permanently disabled, and in the meantime, pay the workman a percentage of his wage to take care of his dependents.

A workman in a classified industry is entitled to compensation in the event of sustaining personal injury by accident arising out of and in the course of his employment. The right of the injured workman to sue his employer is taken away, and the adjudication of his claim taken from the courts to a Compensation Board. The justification for the existence of the Board is that relief to the injured workman and his family shall be prompt and inexpensive, with the elimination of all possible delay and red tape.

Claims for compensation resolve themselves into three main groups: (1) Those obviously compensable. (2) Those obviously not compensable: claims of workmen not covered under the Act and certain fantastic claims with no medical basis whatever. (3) Those where compensability is obscure and doubtful.

* Read at the Seventy-eighth Annual Meeting of the Canadian Medical Association, Section of Industrial Medicine, Winnipeg, June 27, 1947.

Obviously compensable claims are routinely passed and if the disability is evidently of some length the first cheque is mailed at the end of two weeks. However, if the disability period is estimated by the doctor to be less than two weeks, no payment can be made until the doctor notifies us of the date the man is able to return to work. Delay in sending this subsequent report causes delay in payment of compensation and hardship to the workman. Where disability is prolonged, progress reports are necessary as no payment can be made until medical evidence of continued disability is on file.

Claims in group 3 are adjudicated when the evidence on file is as complete as possible; and in this group considerable delay can occur. It is also in this group where most of our misunderstanding occurs; not only with the workmen, but also with the medical profession.

When a workman reports to you with some injury or disease, you naturally take a careful history. The normal attitude of our profession to a history is to assist us to arrive at a diagnosis and thus proceed with treatment. We are not acutely interested in the etiology of the trouble at the moment. However, from the standpoint of compensation, we must accept a different concept of etiology. Compensation administration is interested in establishing a claim: "Did this condition arise by accident out of and in the course of the employment?" Clearly the employer does not want to pay, and cannot be expected to pay for some disease that did not originate in the environment of his industry. Thus the history should be accurate as to time of occurrence, type of injury, evidence of injury at examination, and record all the data accurately. The importance of accurate reports becomes very evident when a case must be reviewed years after the accident.

The history obtained from the patient, together with the findings on examination, should disclose whether the disability complained of is one arising by accident out of his employment. In groups 1 and 2 this will generally be self-evident, but in group 3, where the relationship is not clear there may be considerable difference of opinion. Take for example the following: epididymitis; ganglion; coronary thrombosis; hæmaturia following minor abdominal injury; sarcoma. It would hardly seem, in reviewing the pathology of these lesions that trauma played any part in their etiology, but opinion can be found in the literature supporting this contention. Thus we have frequent claims for these conditions.

Many difficulties arise also in dealing with diseased conditions that either were preceded by, or followed an injury. In the former, estimate must be made of probable aggravation due to trauma, *e.g.*, strain to previously arthritic joints. In the latter the claimant quite honestly believes that the disease which ensues is the direct result of the injury, whether the injury was recent or several years previously: where sequelæ to injury may be due to several causes, there should be a direct sequence or chain of symptoms to establish an etiological relationship in the particular case under review and the weight of probability rather than possibility must be considered.

Workmen are permitted to select their own physician and we endeavour to interfere as little as possible with treatment. We are in favour of well established methods of treatment, and not enthusiastic over any methods which are still in the experimental stage. Permission must be obtained to proceed with elective surgery, and an increasing supervision in these cases can be expected. In Manitoba, where a difference of opinion exists between the Board and the attending surgeon as to elective treatment, the opinion of an independent medical board is obtained. We have found the advice of this board of inestimable value.

Disability ends when the man has recovered and returns to work. However, in many cases complete recovery is not attained and some temporary or permanent disability remains. In these latter cases we endeavour to rehabilitate the man in an occupation within his capacity, and assist him by paying him on a wage loss basis. In a few cases it is necessary to assess a man with an earning capacity and reduce his compensation in order to encourage him to return to some useful employment. Some cases, usually following a minor injury, develop neurotic tendencies; these should be recognized as early as possible and suitable steps taken in treatment. Here the full co-operation between the attending physician and the Board is paramount if success is to be obtained. Experience has taught us that kindly handling of these cases is not always in the best interests of the workman, and although probably old fashioned,

we have found that terminating compensation payments is an effective method of treatment.

Permanent disabilities are estimated when recovery is as complete as possible. Ratings of 10% or less are usually settled in lump sum payments. Those over 10% are paid on a pension basis for life. In Manitoba, we refer a good many of our permanent disability cases to a medical board for independent rating.

INTESTINAL TUBERCULOSIS

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TUBERCULOUS infection of the gastro-intestinal tract is a common disease to those who see and treat large numbers of patients suffering from pulmonary tuberculosis. In general practice, on the other hand, it is rather rarely encountered, and in many instances, the proper methods of treatment are poorly understood. The apparent fear of general surgeons to perform abdominal operations on patients suffering from pulmonary tuberculosis, seems to us to be exaggerated, provided modern facilities are available for proper anæsthesia, etc.

Two rather different types of gastro-intestinal tuberculosis exist, namely, primary and secondary infection. The primary form is an extremely rare disease indeed, and in this country is very seldom seen. The infecting bacillus is usually the bovine type and the lesions are hyperplastic in nature and usually involve the cæcum and ascending colon. They produce symptoms similar to those of constricting carcinoma and often appear like carcinoma even at operation. In these cases, signs of old or recent tuberculosis are not to be found in the lungs.

The second type is the one commonly seen and is always secondary to old or recent, open, pulmonary tuberculosis, and is usually secondary to a pulmonary tuberculosis that some time or other has had a high Gaffky count. The bowel lesions occur as a result of swallowed bacillary sputum and not as a result of hæmatogenous spread, as supposed by some authors. The initial lesions of secondary tuberculosis are ulcerative, but many of these progress to a sclerotic and constricting type of lesion.

The complication has a serious prognosis. Hippocrates long ago stated that phthisical persons die if diarrhœa sets in. That was about universally true in his day, but fortunately is not wholly so today. Intestinal tuberculosis is the commonest complication of pulmonary tuberculosis. Just how frequent it is among our own patients I am not prepared to say as we do not do routine x-ray examinations of the gastro-intestinal tract. The various autopsy figures range from 50 to 80%, but this, of course, does not give the true incidence as occurring in all cases of pulmonary tuberculosis. The incidence in various sanatoria would vary a great deal anyway, depending upon the percentage of minimal, moderately advanced, and far advanced, cases admitted.

It is probably safe to say that at least 90% of our own hopeless, far advanced, open cases, unfit for collapse therapy, have extensive tuberculous intestinal lesions. Blumberg reports x-ray evidence of bowel tuberculosis in 5 to 8% of minimal cases, 14 to 18% of moderately advanced, and 70 to 80% of far advanced. Improvements in x-ray technique since the publication of his paper would doubtless raise the incidence.

The stomach itself is rarely involved. At our institution we have not yet found a case proved to have gastric tuberculosis, although some may have been missed due to the fact that we have not done routine gastrointestinal x-rays and that, although our autopsy rate is reasonably high, it still leaves much to be desired. In an institution such as ours, serving such a wide area, routine autopsies are hard to obtain. About 85% (Bockus) of intestinal tuberculosis affects the ileo-cæcal region and both the frequency and intensity of the disease diminishes both proximal and distal to this point. This certainly has been our experience in cases operated upon and examined at autopsy.

The initial lesions are to be found in the lymphoid patches of the gut and in the appendix, itself a lymphoid organ. This latter fact is of extreme clinical significance in dealing with appendicitis in the tuberculous patient as will later be pointed out. The typical round and giant cell infiltrations first occur and later, caseation and ulceration. This active stage may exist until death unless the pulmonary disease

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is controlled, or coincident with control of pulmonary disease it may itself "heal" by the formation of hyperplastic, and in many instances, constricting, obstructing fibrous lesions. In some cases the bowel tuberculosis does not progress beyond the ulcerative stage even with good control of the pulmonary lesion. When a patient is not progressing satisfactorily after control of lung disease, ulcerative bowel tuberculosis must always be suspected and if proved to exist, treated in an active manner.

SYMPTOMS

The symptoms of bowel tuberculosis are extremely varied and no orderly sequence of symptoms and signs can be enumerated as can be with most inflammatory conditions of the gastro-intestinal tract. In the first place the picture is confused by the fact that a large number of patients with pulmonary tuberculosis complain of gastro-intestinal symptoms when no gastro-intestinal lesion exists at all. Anorexia, nausea and vomiting are rather common symptoms complained of in straight uncomplicated pulmonary tuberculosis. Bouts of epidemic diarrhœa are relatively common in tuberculous institutions.

The patient, however, who complains of constant abdominal pain, usually has bowel tuberculosis. This pain may vary in character and site. In our experience the history is that the pain was at first, vague and generalized and as time progressed became more localized in the right lower quadrant. It is often made worse by eating and relieved somewhat by a bowel movement. Anorexia and nausea are rather common symptoms. The fact that symptoms are produced that have a profound effect on the nutrition of the patient, and consequently on his cure, has possibly led us to adopt an attitude of more radical and immediate surgical intervention rather than the slower and probably, more uncertain, conservative, measures commonly advocated. When the lesion progresses to the sclerotic stage, the pain becomes cramp-like and spasmodic and symptoms and signs of partial or complete bowel obstruction make their appearance.

The onset of diarrhœa in the tuberculous patient should arouse suspicion of the presence of bowel tuberculosis. This is a common symptom of the complication. It is usually intermittent, but in advanced cases may be constant. Hæmorrhage from the ulcerations is seldom seen due to the fact that early in the disease a condition of endarteritis obliterans is set up around the ulcerated areas.

Ordinary physical examination may reveal no abnormal findings unless the peritoneum is involved or obstruction is present. Most frequently, however, these patients complain of slight to marked tenderness in the right lower quadrant.

DIAGNOSIS

X-ray studies afford the only accurate means of diagnosis of intestinal tuberculosis. Brown and Sampson of Saranac Lake (quoting Bockus) state that the x-ray signs include: (1) general intestinal hypermotility with complete or nearly complete emptying of the colon in twenty-four hours; (2) failure of the cæcum or ascending colon to retain barium (Stierlin sign); (3) the presence of spastic filling defects and absence of haustrations in the ascending colon; (4) segmentation of coils of small bowel with or without distension of small bowel loops; (5) abnormal retention of barium in the ileal loops and gastric retention. In advanced cases with fixation of small bowel loops very marked distortions and angulations are to be noted. The barium enema in advanced cases often exhibits most of the characteristics of ordinary ulcerative colitis with the exception that in tuberculosis the greatest amount of disease is seen in the cæcum and ascending colon. In cases with partial obstruction the diagnosis is frequently easily made by the use of flat plates alone without the use of barium contrast media.

The diagnosis, of course, is made on the clinical picture and x-ray studies. The finding of tubercle bacilli in the stools is of little diagnostic value as they may be found in cases of pulmonary tuberculosis uncomplicated by lesions in the bowel. It should be mentioned here perhaps that patients with controlled lung disease who continue to run fever and are toxic, most probably have active bowel tuberculosis.

In regard to treatment the majority of authors feel that it should be conservative in so far as possible. They advocate bland diet with vitamin supplements particularly vitamin C and B. In the presence of diarrhœa the diet should be low fat and high carbohydrate and protein. In the presence of diarrhœa, calcium loss is excessive and this should be supplemented.

TABLE I.

Age	*Sex*	*Stage*	*T.B.*	*Symptoms*	*Signs*	*Operation*	*Pathology*	*Anæsthesia*	*Healing*	*Complications*	*Outcome*	*Cause of death*
16	M.	F.A.	+	Pain in R.L.Q. Nausea. 36 hrs.	Slight Tend. R.L.Q.	Appendectomy Mod. inflamed.	Numerous tbc. follicles in submucosa	Pont.	Primary	Nil	Recovery. Discharged arrest.	
34	F.	M.A.	O	Pain severe. R.L.Q. becoming general. 72 hours.	Slight tend. R.L.Q	Appendectomy. App. and lower cæcum mod. infl.	Typic. tbc. app. Numerous follicles with giant cells in submucosa.	Pont.	Primary	Nil	Recovery. App. cured	
35	M.	F.A.	+	Chronic right side paid of 2 yrs. dur. Constip. and diarr. Nausea frequent.	Tender in R.L.Q. Slight Ba. enema suggest. Tbc. cæcum.	Appendectomy. Cæcum thickened. App. markedly infl. and swollen.	Few follicles. Very slight tbc. involved.	Nuper.	Primary	Nil	Improved. Still mod. pain	
25	F.	M.A.	+	Moderate R.L.Q. pain. Nausea. 24 hours.	Mod. tender R.L.Q.	Appendectomy. Mod. inflamed.	Some small typical tbc. follicles in submucosa.	Nuper.	Primary	Nil	Recovery arrested	
22	F.	F.A.	+	Acute severe pain general; marked in R.L.Q. Nausea, vomit. 18 hours.	Markedly tender R.L.Q.	Appendectomy. Mod. swollen and inflamed.	Simple subacute. Few tbc. follicles in submucosa.	Pont.	Primary	Tuberculous peritonitis	Died 6 mos. postop.	Pulmonary? Peritonitis?
30	M.	F.A.	+	Slight to mod. severe pain in R.L.Q. Nausea. 24 hours.	Slight tend. R.L.Q.	Appendectomy. Slightly inflamed.	Subacute. Few tbc. follicles in submucosa.	Nuper.	Primary.	Nil	Died	Operative thoracoplasty
33	F.	F.A.	+	Numerous attacks mod. pain R.L.Q. Nausea, constipat'n frequent. 6 mos. duration.	Slight tend. R.L.Q. Pelvic o.k.	Appendectomy. App. long small sclerosed.	Few follicles in submucosa.	Novoc.	Primary	Nil	Died	Pulmonary tb. 3 yrs. postop.
27	F.	F.A.	+	Slight pain. No nausea. 10 hrs.	No tend. Mod. guarding R.L.Q.	Appendectomy. Marked inflamed and thickened.	Tb. of wall of ap-appendix.	Pont.	Primary	Tuberculous	Died. 6 mos.	Tbc. peritonitis.
20	F.	F.A.	+	Mod. pain in R.L.Q. Nausea 48 hrs.	Mod. tend. R.L.Q.	Appendectomy. Mod. inflamed.	Many typical follic. and giant cells in all layers.	Av. N20	Primary	Nil	Died 2 yrs.	Pulmonary tb.

TABLE I.—*Continued.*

Age	*Sex*	*Stage*	*T.B.*	*Symptoms*	*Signs*	*Operation*	*Pathology*	*Anæsthesia*	*Healing*	*Complications*	*Outcome*	*Cause of death*
47	M.	F.A.	+	Severe pain in R.L.Q. No nausea.	Marked tend. with guarding R.L.Q. 24 hrs. 1st attack.	Appendectomy. Appendix mod. inflamed.	Simple subacute with follicles in submucosa.	Pont.	Primary	Nil	Recovery	
29	F.	F.A.	+	Slight pain in R.L.Q. 1 day.	Mod. tend. R.L.Q.	Appendectomy. Mod. inflamed.	Tbc. follicles in submucosa.	Novoc.	Primary	Nil	Died 8 yrs. postop.	Pulmonary tb.
28	F.	F.A.	+	Mod. pain R.L.Q. 4 days.	Slight tend. R.L.Q. Guarding.	Appendectomy. Mod. inflamed.	Simple acute diffuse. Few tbc. follicles in submucosa.	Pont.	Primary	Nil	Recovery arrested	
32	F.	M.A.	O	Pain in R.L.Q. 48 hrs.	Slight tend. R.L.Q.	Appendectomy. Slightly inflamed.	Mod. tbc. involv. of all coats	Nuper.	Primary	Nil	Died 1 year, postop.	Pul. tb.
29	F.	F.A.	+	Pain in R.L.Q. Nausea. 2 days	Mod. tend. R.L.Q.	Appendectomy. Mod. thick and inflamed.	Chronic tbc. appendix with acute superimposed changes.	Nuper.	Primary	Nil	Recovery arrested	
27	F.	F.A.	+	Nausea. Aching pain in R.L.Q. 36 hrs.	Marked tend. Rigid. R.L.Q.	Marked thick and infl. app. Obvious disease cæcum.	Acute supp. with many tbc. follicles in wall.	Nuper.	Primary	Nil	Recovery arrested	
21	F.	F.A.	+	Several attacks pain in R.L.Q. c nausea, vomit.	Mod. tend. R.L.Q.	Appendectomy. Slightly inflamed.	Mild acute diffuse tbc. appendicitis	Pont.	Primary	Nil	Recovery arrested	
18	F.	F.A.	+	Pain in R.L.Q. 24 hrs. nausea.	Mark. tend. R.L.Q.	App. greatly swollen retrocæc., infl. Cæcum thick. and œd.	Supp. append. Gran. tissue. Typic. serosa follicles.	Novoc.	Drain. 3 mos.	Nil	Unimproved	
26	F.	F.A.	+	Pain in R.L.Q. 36 hrs. No vomit.	Mod. tend. R.L.Q.	Appendectomy. App. mod. infl.	Num. follic. with giant cells submucosa.	Novoc.	Primary	Nil	Recovery arrested	
17	F.	M.S.	+	Sev. gen. abd. 48 hrs. Loc. to R.L.Q.	Mod. tend. R.L.Q.	Appendectomy. App. med. infl. and swollen tip.	Small tbc. ulceration in mucosa.	Nuper.	Primary	Nil	Recovery arrested	
28	F.	F.A.	+	Mod. pain in R.L.Q. 2 days. Nausea.	Mod. tend. R.L.Q.	Appendectomy. Great thick. infl. Cæcum nodular. Pelvic tbc. peritonitis.	Tbc. appendix. Numerous follicles in submucosa.	Pont.	Primary	Nil	Died 6 mos. postoperative	Pul. tuberculosis.

TABLE I.—*Continued.*

Age	*Sex*	*Stage*	*T.B.*	*Symptoms*	*Signs*	*X-ray*	*Operation*	*Pathology*	*Healing of incision*	*Complications*	*Result*	*Cause of death*
36	M.	F.A.	+	Severe abd. pain 2 yrs. previous. App. removed. Tbc. entercolitis diag. Subsequent attacks mild. Progressive cons. pain, vomit. 2 weeks.	Distension. Fullness and tender mass in R.L.Q.	Flat films reveal small bowel fluid levels. Ladder pattern.	Complete obstruation at ileo-cæcal valve. Resection of ascend. colon, cæcum, 2½ ft. of ileum. Side - to - side terminal small bowel with transverse colon. No drainage.	Tbc. stricture at ileo-cæcal valve.	Primary	Nil	Recovery 2½ yrs.	
23	M.	F.A.	+	Attacks of R.L.Q. pain 18 mos. Present attack much more severe. 2 wks. Crampy pain nausea, vomit. No diarr. Loss of wt.	Moderate tenderness R.L.Q. No mass.	Stomach and Duodenum, Ba. meal. Stomach very long and ptosed. No ulcer Small and large bowel not examined.	High midline incision. Appendix cæcum ascend. colon terminal ileum thick and inflamed. Tabes mesenterica. Resect. ascend. colon. cæcum and app., 18″ ileum. Side-to-side transverse colon to ileum. No drain.	Hyperplastic and early ulcerative tb. of bowel, and append. Tabes mesenterica.	Primary	Nil	Excellent. Relief of all symptoms. Gain of 40 lbs. 18 mos.	
19	M.	M.A.	+	Pain in R.L.Q. of 8 hrs. History of previous attack. Nausea and vomiting.	Marked tenderness in R.L.Q. No mass.	Not done. Appeared straight case of acute appendicitis.	McBurney. App. thick and infl. Cæcum solid mass c obst. at ileo-cæcal valve. Thick asc. colon. Res. of asc., cæc., term. ileum. Side-to-side ileum to trans. No drain.	Hyperplastic ulc. of cæcum and colon. Inv. of mesenteric glands.	Infection. Drained for 3 wks.	Nil	Complete relief of symptoms Tb. app. cured.	
20	F.	F.A.	+	4 months prev. perforat. append. removed. Tbc. typhlitis seen at op. App. symptoms of int. obstruction.	No record.	Upright flat fluid levels. Supine ladder pattern. Small bowel.	Rt. Rectus. 5 tbc. strictures last 4 ft. of ileum with complete obst. Resection of app. 4½ ft. of ileum c side-to-side ileum to ileum. No drain.	Typical fibro-caseous tbc. ulc. with numerous strictures	Infection. Persistent sinuses.	Subhepatic abscess drained one week later.	Death. 4 years later.	Pul. tb.
53	M.	F.A.	+	2 months repeated attacks of disten. pain, vomit. Good results from all enemas.	No record.	Complete G.I. series. Constriction in mid portion of ascending colon. Malignancy.	Right rectus. Hard mass in cæcum. Malignant? Resection of ascend. colon, cæc., 8″ term. ileum. Side-to-end, small to large. No drain.	Typical tb. cæcum and ileo-cæc. valve. Glands involved.	? ? ?	Paralytic ileus.	Death 7th post-op. day.	Paralytic ileus.

TREATMENT

It would appear to us that no set rule of treatment can be applied, but rather that treatment must be selected suitable to the combination of conditions present in the particular case under study. In the first place, accepting that the bowel lesion is the result of swallowed bacillary sputum, the first step in the control of the bowel lesion is to control the lung disease and render the sputum negative. This should be done at the earliest possible moment. In these particular cases compression therapy finds a wide field of usefulness. There should be no delay in the application of pneumothorax, thoracoplasty etc., if considered suitable for the lung lesion, even in the presence of fever and toxic symptoms. If such measures are promptly taken and are effective, the majority of early ulcerative, and in some cases extensive ones, will heal by themselves with the help of diet and vitamin therapy. In the event of bowel lesions not healing with controlled lung disease, we feel that the intestinal lesion is best treated by resection rather than prolonged medical treatment, provided it is sufficiently localized to allow this procedure. Even extensive resections are usually well tolerated and subsequent bowel function good. When obstructive lesions are present, the treatment must be surgical.

Most confusion seems to exist in the minds of physicians and general surgeons in cases, where for one reason or another, the lung condition cannot be immediately controlled, and in which digestive disturbances exist along with tenderness in the right lower quadrant. We have seen patients with unstable lung disease, in whom a diagnosis of chronic appendicitis has been made, and operation advised against simply because they had pulmonary tuberculosis, and the fear that they might have a tuberculous appendix. We feel that this attitude is definitely wrong and that the direct opposite is correct. Chronic intestinal disease with resultant disturbed function and malnutrition is bound to lower the resistance of a tuberculous patient, and retard or prevent satisfactory progress in the lung. If the disturbing abdominal lesion is removed, the general and local lung improvement is often remarkable. Such treatment, of course, is not indicated in cases where the lung disease itself renders a favourable outlook hopeless.

In a series of 109 consecutive cases operated upon for acute or subacute abdominal conditions, 101 suggested the diagnosis of acute appendicitis and 8 had evidence of obstructive lesions. Of the 101 presenting symptoms of appendicitis 20 or 19.8% were found to have tuberculosis of the appendix or ileo-cæcal region and 81 or 80.2% were found to be simple acute appendicitis without pathological evidence of tuberculosis of the appendix. The 20 localized tuberculous cases were treated by simple appendectomy or added partial resection of the cæcum. Of this series 8 presented evidence of more extensive and obstructing tuberculosis and were treated by radical bowel resections. All 8 were diagnosed as tuberculosis but only 5 proved to be so. The pathological report on one was carcinoma and regional ileitis, non-tuberculous, on two others. Twenty-five cases or 22.9% of the series of 109 were tuberculous. Of the 25 tuberculous cases, 20 or 80% had far advanced lung disease and 5 or 20% had moderately advanced disease at the time of their operation. None were classified as minimal. Twenty-three or 92% had positive sputum at the time of operation while 2 or 8% were negative, but had had positive sputum in the past.

Table I is a summary of the cases operated upon.

CONCLUSIONS

1. Intestinal tuberculosis is a common disease in tuberculosis institutions.
2. It occurs as a direct result of swallowed bacillary sputum.
3. The first and most important step in its control and treatment is control of lung disease.
4. Control of lung disease will effect cure of ulcerative bowel tuberculosis in a large percentage of cases.
5. The results of surgical treatment of intestinal tuberculosis are satisfactory.
6. A small series of cases is presented.

You may derive thoughts from others; your way of thinking, the mould in which your thoughts are cast, must be your own. Intellect may be imparted, but not each man's intellectual fame.—Charles Lamb.

MINIATURE CHEST X-RAYS

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THE use of miniature chest x-rays to detect those persons requiring further study for intrathoracic disease is now sufficiently established to warrant a brief review of the principles involved. It is generally agreed that periodical chest x-rays of "apparently healthy" individuals will reveal unsuspected intrathoracic disease and abnormalities requiring further observation and investigation. A proper understanding of the methods and results in this work is of interest to all physicians.

TECHNIQUE

While a photofluorographic examination of the chest is a type of x-ray examination, it should be recognized that a "minature chest x-ray" is actually not a radiograph but a photograph upon transparent film of an image which is momentarily induced upon a florescent screen. In fluoroscopy we see the image on the fluorescent screen using x-ray equipment of low milliamperage and a coarse-grain screen. In photofluorography we use a special high-speed camera to photograph the image using high-powered x-ray equipment and a fine-grain, supersensitive fluorescent screen enclosed in a light-proof hood. The x-rays may be generated by any standard x-ray equipment of sufficient capacity. The light-proof hood containing stationary grid, fluorescent screen and camera may be arranged in the x-ray room so that chest photofluorography may be performed, in addition to conventional radiography, utilizing the same x-ray generating equipment, merely by rotating the x-ray tube from one position to another. Preferably a photo electric timer should be used to provide uniformity and to eliminate the necessity to adjust kilovoltage and exposure time according to the thickness of the chest. Recent practice in rapid screening by chest photofluorography makes complete disrobing of the chest unnecessary. Subjects are requested to remove clothing over the chest down to shirt or blouse and to remove any large metallic objects from the clothing remaining over the chest. Buttons are usually in the mid-line but if not, are easily recognized as such. If difficulty in interpretation is encountered, the subject is recalled for further examination.

The Gage Institute in Toronto, operated by the National Sanitarium Association, conducts chest x-ray surveys in communities and large industries using transportable x-ray equipment taking single exposures on 70 mm. roll film.

ACCURACY FOR SCREENING

Several studies have been made in an attempt to assess the accuracy or sensitivity of miniature chest x-rays, the criterion of sensitivity usually being the ability to demonstrate lesions of pulmonary tuberculosis, particularly in the minimal stage.

A Board of Roentgenology[11] appointed by the United States Department of Veterans' Affairs made a detailed comparison of the effectiveness of various radiographic and photofluorographic methods in tuberculous case-finding. A statistical analysis was made of the reports from two experienced radiologists and three tuberculosis specialists upon four types of chest x-ray examination upon each of 1,256 subjects (single 14" x 17" celluloid films; single 14" x 17" paper sheets; stereoscopic 4" x 5" photofluorographs; and single 35 mm. photofluorographs). The five physicians arranged preliminary agreement upon criteria for classification but had no recourse to the opinion of any other and no means of identifying the various types of examination with any subject. The conclusions of this detailed study are "except for a slight disadvantage to the miniature technique resulting from over-reading, not one of the methods, not even the 14" by 17" celluloid radiographic films, is superior, for case finding purposes, to any of the other methods". In the opinion of that Board, such an advantage as may be inherent in any one technique is of so small a magnitude that it is very much smaller than the human error involved in x-ray interpretation.

Plunkett, Weber and Katz[7] studied the relative accuracy of single 4" x 5" films and single 35 mm. films to show lesions of minimal pulmonary tuberculosis as compared with 14" x 17" films. They state, "by using the 4" x 5" film, 98.3% of all cases of clinically significant tuberculosis would have been found; while by using the 35 mm. film, 91.8% of the cases would have been diagnosed. Both miniature methods were 100% accurate in diagnosing moderately advanced and far advanced cases."

In our opinion, it is doubtful if we should attempt without qualification to assign any graded accuracy to the various methods of routine chest x-ray examination for the reason that perhaps more important than the *method* of x-ray examination is the *carefulness* and *skill* with which the method is used. It will be appreciated that even stereoscopic 14" x 17" radiographs of the chest of poor technical quality or carelessly interpreted may be useless or even dangerous. Assuming, however, that technical factors are equal and that the x-ray films are interpreted by careful physicians with experience, it is the opinion of the authors that photo-

fluorographs obtainable to this date will indicate the *presence* of opacities, but provide less detail for diagnostic study than 14″ x 17″ radiographs. Such slight loss of detail might conceivably result in non-portrayal of extremely early parenchymal lesions of little density. Here, of course, the error is of such low magnitude that it is quite overshadowed by the variation in judgment as to what falls within normal limits among different well-qualified readers, or of the same interpreter at different times. As in radiography, detail and contrast are more difficult to obtain in subjects with extremely thick chests. Among miniature chest x-ray films the greater detail is found in 4″ x 5″ films with 70 mm. film following a close second. No opinion can be expressed upon 35 mm. film due to lack of sufficient experience.

For any given method of chest x-ray examination, two exposures upon the same subject with the x-ray tube in two positions and the two films read singly or stereoscopically decrease the chance of error. Thus, stereoscopic 4″ x 5″ miniature films could reveal a small area of decreased radio-translucency which might be missed in a single 14″ x 17″ radiograph.

It should be understood that although good photofluorographs properly interpreted will demonstrate the *presence* of variations from the normal, clinical study and evaluation is required to establish a diagnosis of disease. In the study of those screened by the "miniature chest x-rays", the clinician will usually require x-ray examinations providing greater detail and in certain cases with views at various angles, such as lateral and oblique, etc.

USEFULNESS

It is recognized that "looking into the chest" by *any* type of x-ray examination will reveal unsuspected intrathoracic disease not detectable by the usual methods of physical examination. It is equally evident that even the most elaborate and complete type of chest x-ray examination would require clinical interpretation and judgment by those experienced in diseases of the chest for those subjects showing significant abnormalities in the x-ray films. The problem therefore resolves itself into selecting some satisfactory and relatively inexpensive method of screening all individuals to detect those requiring further study, clinically and radiologically. Chest photofluorography has provided that method. Although most work with miniature chest x-rays has been primarily for the detection of "early" asymptomatic pulmonary tuberculosis, a significant number of non-tuberculous abnormalities and diseases have been found, the detection of which constitutes an important "by-product" of the survey. Such screening by chest photofluorography finds its place in:

(*a*) Periodic chest x-ray surveys of the general population by communities, and by large industries as part of the anti-tuberculosis campaign.

(*b*) Routine screening of all hospital admissions and out-patients to protect hospital personnel and other patients from tuberculous infection as well as to detect unsuspected non-tuberculous pulmonary disease requiring attention.

(*c*) Routine screening of all new employees in industry as part of the "pre-employment" or "on employment" examination.

(*d*) Periodic chest x-ray surveys of persons in institutions, schools, universities, mental hospitals, etc.

(*e*) Periodic chest x-ray examination of special groups such as hospital personnel, food handlers, barbers, teachers, etc.

The type and size of miniature chest x-ray to be used will be the subject of consideration along the following lines:

(*a*) For those organizations anticipating only a moderate flow of subjects (less than 100 per day), the single or stereoscopic cut film photofluorograph is to be preferred. These cut films lend themselves to individual and immediate processing. Such cut film for photofluorography is commonly 4″ x 5″ in size for flat, or 4″ x 10″ for stereoscopic exposures. Recently, cut film holders have been provided to substitute for the roll film magazines on the 70 mm. cameras, thus permitting 70 mm. single or stereoscopic exposures on cut film of that size.

(*b*) For those organizations which plan to provide miniature chest x-ray surveys by communities and industries where the flow of subjects will be rapid and continuous, the roll film method using automatic cameras, photo-electric timers, various safety relays and signalling devices, is the method of choice. This equipment will permit up to 100 exposures per hour running continuously for as many as 1,200 exposures per day. Processing and interpreting are carried out upon full length rolls of 100 feet of film containing approximately 400 exposures.

LIMITATIONS

The limitations of photofluorography in screening for intrathoracic disease are actually the limitations of any type of routine chest x-ray examination. Obviously, in such routine chest screening of large numbers of subjects, only the usual P.A. (postero-anterior) views can be expected. Furthermore, when x-ray equipment and technicians are engaged in "mass production" methods, the quality of the product under circumstances of fatigue, carelessness, or technical difficulties may fluctuate within a range determined by the alertness of the chief x-ray technician. There should be a constant desire

and striving for better quality by all members of the technical staff. The prompt processing and viewing of the films will do much to detect unfavourable tendencies, permitting immediate corrective steps.

The careful and scientific physician will always appreciate the limitations of any method of examination which he is employing. A routine P.A. chest x-ray examination will not necessarily disclose evidence of such conditions as: asthma, chronic bronchitis, bronchiectasis, parenchymal opacities posterior to the heart or in the extreme lung bases below the dome of the diaphragm, bronchogenic carcinoma before the atelectatic or hilar enlargement stage, etc. It becomes important therefore to give careful and proper information to those who receive this type of examination.

In the surveys conducted by the Gage Institute, each person x-rayed is given a stub to retain showing the following information in addition to the project number and x-ray number for future reference:

"You have received a miniature chest x-ray for the detection of tuberculosis of the lung.

If abnormal, you will be notified within 10 days.

If normal, you will NOT be notified. If you have symptoms referable to your chest, consult your physician regarding the possibility of chest disease other than tuberculosis.

Even if normal, you may at some future date develop tuberculosis. It is desirable for all adults to have periodic chest x-rays."

Periodic chest x-ray examinations (whether by radiography or by photofluorograph) will detect re-infection type tuberculosis in the minimal stage frequently before symptoms appear and usually before the disease becomes "open". One must appreciate, however, that primary or "childhood" type tuberculosis, including pleurisy with effusion, may develop rapidly. In a very small number of cases, possibly because of massive infection or diminished immunity, the primary stage passes quickly into a progressive re-infection type of tuberculosis with development of cavity formation or miliary tuberculosis.

Among 121,588 persons whose miniature chest x-ray films were considered to show no demonstrable disease in 1946, we have knowledge of 7 persons who were subsequently found to have developed pulmonary tuberculosis up to May 1, 1947. Careful review showed that in only 1 of these cases (G.K.) was an opacity missed in the miniature film. This small opacity was basal and partially obscured by basal lung markings. The details in these cases are as follows:

Initials	*Age*	*Later diagnosis made upon*	*Interval between original miniature film and later diagnosis months*
G.K.	20	Symptoms; later x-ray; Pos. sputum	3
A.A.	17	later x-ray; Pos. sputum	3
C.W.	38	Symptoms; later x-ray	5
A.R.	19	later x-ray; Pos. sputum	5
I.G.	25	later x-ray	6
M.G.	54	Contact; later x-ray	10
P.T.	16	later x-ray	12

As we have evidence that an increasing number of persons are reaching adult life without seeding by the tubercle bacilli, we might therefore expect to see the development of primary type pulmonary tuberculosis among adults more frequently than in the past. In the community surveys, conducted by the Gage Institute, pre-school children are not x-rayed. If an adult is found to have tuberculosis, any pre-school children in that home would of course be adequately examined along with other contacts.

Obviously, certain individuals will fail to take advantage of the free periodic chest x-rays, and will await symptoms or definite illness before consulting a physician. By that time, moderate to far advanced pulmonary tuberculosis is frequently present and the patient has been a source of infection to others. Therefore, the Gage Institute not only conducts periodic chest x-ray surveys among apparently healthy individuals but also maintains a chest clinic at headquarters where facilities are provided for the examination not only of those found to require such in the x-ray surveys, but also of those persons referred because of symptoms, because of contact with tuberculosis, or as part of post-sanatorium care. (The Gage Institute Chest Clinic examinations in 1946, exclusive of those referred because of abnormal miniature films, yielded active pulmonary tuberculosis in 2.4% of the examinations. This yield was 22 times greater than the incidence of active pul-

monary tuberculosis found in the miniature chest x-ray surveys of apparently healthy persons.)

Tuberculosis case-finding by chest photofluorography of apparently well persons should be considered as a valuable addition to, but should not supplant, the provision of free chest clinics and private chest consultation.

RESULTS

The literature contains a considerable number of reports upon the results of miniature chest x-ray surveys among certain groups, including members of the Armed Forces in various countries. The results of such surveys will vary according to:

(*a*) The age group and sex of those under study. (*b*) The degree of "tuberculinization" of the population from which the group surveyed is selected (as may be deduced from the results of tuberculin surveys or the tuberculosis mortality rate in the general population). (*c*) The prime interest or main purpose of the survey (*i.e.*, to detect and report mainly gross tuberculosis or to include slight abnormalities and non-tuberculous abnormalities). (*d*) The criteria accepted by the interpreters in respect to what findings are considered as within or beyond normal limits. (*e*) Whether the published results are based solely upon miniature x-ray findings or whether such are reported only after clinical study of those screened out by the survey. Results will also vary according to the extent and detail of the clinical investigations, and whether the reported diagnoses take into consideration the sputum reports which may be delayed (*i.e.*, for tubercle bacilli on culture).

Our subjects were practically all white and lived in southern Ontario (mainly in Toronto and suburbs) where the tuberculosis mortality rate for 1945 was 28.5 per 100,000. Many subjects in this district had received previous routine chest x-rays in the armed forces or in industrial chest x-ray surveys within the previous five years. The miniature chest x-rays are classified, and the clinic examinations conducted by our group of four full-time physicians who are in constant collaboration with each other. Our interest is in the detection of any significant intrathoracic disease. Those persons with no disease demonstrable in the miniature films are not notified. Those with minor abnormalities are notified by letter and advised to consults their physicians and have repeat chest x-rays at stated intervals. (This procedure is also followed in a very small number of cases presenting evidence in the miniature chest x-ray indicating minimal pulmonary tuberculosis where the opacities are apical and radiologically "hard"). Those with obvious non-tuberculous

TABLE I.

SUMMARY OF RESULTS OF SCREENING FOR INTRATHORACIC DISEASE BY MINIATURE CHEST X-RAYS AMONG 126,089 APPARENTLY HEALTHY PERSONS BY THE GAGE INSTITUTE, IN 1946

	No.	*% of group*	*% of total x-rayed*
Pulmonary tuberculosis, active (including pleurisy with effusion)			
Pleurisy with effusion	5	3.7%	.....
Primary type	1	0.8%	.....
Re-infection type—Minimal (Pos. sputum in 7)	64	48.1%	.....
—Mod. Adv. (Pos. sputum in 16)	48	36.1%	.....
—Advanced (Pos. sputum in 7)	15	11.3%	.....
TOTAL (Pos. sputum in 30)	133	100%	0.11%
Pulmonary tuberculosis, inactive (including suspected tuberculosis; also apical scarring as pulmonary tuberculosis, minimal, inactive)			
Suspected pulmonary tuberculosis	66	6.5%	.....
Re-infection type—Minimal	800	79.1%	.....
—Mod. Adv.	132	13.1%	.....
—Advanced	13	1.3%	.....
TOTAL	1,011	100%	0.80%
Non-pulmonary tuberculosis	9	.....	.....
TOTAL TUBERCULOSIS (all forms) (active and inactive)	1,153	.....	0.91%
Non-tuberculous diseases or abnormalities	777	.....	0.61%
Diagnosis deferred or undiagnosed	131	.....	0.10%
Accentuated lung markings	181	.....	0.14%
Previous pleurisy	830	.....	0.65%
Pulmonary calcifications	1,146	.....	0.97%
Film technically unsatisfactory or not taken	283	.....	0.22%
TOTAL (all abnormalities)	4,501	.....	3.6 %
No disease demonstrable	121,588	.....	96.4 %
GRAND TOTAL	126,089	.....	100%

disease are notified by letter and advised to consult their private physicians for further investigation. Those with abnormalities which suggest or can not be differentiated from tuberculosis are notified and given appointments for clinic examinations at the centrally located Gage Institute Chest Clinic. Following that examination, a complete report is sent to the private physician. When tuberculosis or suspected tuberculosis is confirmed, a copy of the report is sent to the medical officer of health of the municipality in which the patient lives. When any individual fails to report as advised, notification is made to the medical officer of health who arranges for a visit by a public health nurse who is usually able to persuade the individual to report to the Gage Institute for examination. If the examination is performed at some other chest clinic or by private chest consultant, the results are obtained by the public health nurse and forwarded to the Gage Institute.

The statistical cards (Hollerith punch card system) for those recalled for clinic examination are not completed until at least three months after the miniature films are taken. To that extent, the statistics reflect the results of sputum examinations arranged at the initial clinic examination, or the result of a second examination when advised within that period. Having reported to the clinic for examination because of an abnormal miniature chest x-ray, the person is then subject to the regular clinic follow-up procedure, for periodic recall if and when advised.

Table I shows a summary of the findings among 126,089 apparently healthy individuals provided with miniature chest x-rays in 1946 by the Gage Institute. Table II shows the actual

TABLE II.

ACTUAL PERCENTAGES OF ACTIVE AND INACTIVE PULMONARY TUBERCULOSIS ACCORDING TO AGE GROUP AND SEX (GAGE INSTITUTE)

Sex	*Age groups*	*No. x-rayed*	*Active Pulm. Tbc. (including pleurisy with effusion)*		*Inactive pulmonary tuberculosis*		*Active and inactive pulmonary tuberculosis*	
			No.	*%*	*No.*	*%*	*No.*	*%*
Male	0-4 yrs.	313	0	—	0	—	0	—
	5-9 "	5,340	0	—	1	0.02	1	0.02
	10-14 "	9,123	2	0.02	4	0.04	6	0.07
	15-19 "	13,763	10	0.07	10	0.07	20	0.15
	20-24 "	4,332	11	0.25	8	0.18	19	0.44
	25-29 "	3,545	4	0.11	24	0.68	28	0.79
	30-39 "	7,779	5	0.06	62	0.79	67	0.86
	40-49 "	7,117	10	0.14	131	1.8	141	2.0
	50-59 "	4,843	14	0.29	173	3.6	187	3.9
	60-69 "	2,435	8	0.33	99	4.1	107	4.4
	70 & over	541	1	0.18	31	5.7	32	5.9
	not stated	1,513	2	0.13	19	1.3	21	1.4
Female	0-4 yrs.	286	0	—	0	—	0	—
	5-9 "	5,086	0	—	0	—	0	—
	10-14 "	9,151	5	0.05	2	0.02	7	0.08
	15-19 "	14,276	12	0.08	13	0.09	25	0.18
	20-24 "	6,707	14	0.21	25	0.37	39	0.58
	25-29 "	5,068	10	0.20	27	0.53	37	0.73
	30-39 "	9,997	13	0.13	98	0.98	111	1.1
	40-49 "	6,768	4	0.06	101	1.5	105	1.6
	50-59 "	3,677	3	0.08	79	2.1	82	2.2
	60-69 "	1,683	0	—	66	3.9	66	3.9
	70 & over	492	1	0.20	22	4.5	23	4.7
	not stated	2,254	4	0.18	16	0.71	20	0.89
Male	Total	60,644	67	0.11	562	0.93	629	1.0
Female	Total	65,445	66	0.10	449	0.69	515	0.79
TOTAL		126,089	133	0.11	1,011	0.80	1,144	0.91

Under "inactive pulmonary tuberculosis" are included suspected pulmonary tuberculosis, apical scarring, and pulmonary tuberculosis of questionable activity, as well as definitely inactive.

percentages of active and inactive pulmonary tuberculosis according to age group and sex.

SUMMARY

1. All other factors such as care and skill being equal, photofluorography compares favourably in sensitivity with 14" x 17" radiographs for screening purposes (*i.e.*, the detection of abnormalities for further study).

2. Routine chest x-ray examinations will not necessarily reveal evidence of certain non-tuberculous diseases and will not forecast the subsequent development of pulmonary tuberculosis in those who are classified as "negative" upon a single examination.

3. In our experience, 2% of those receiving miniature chest x-ray films required further examination for possible tuberculous disease. Approximately 30% of that group proved to have no demonstrable disease. The percentage recalled for further examination and to some extent the amount of "over-reading" from the miniature films will be greatest when interpretation is over-cautious, when any clothing remains on the chest, when films are technically substandard and when the older age groups are being screened.

4. Among 126,089 miniature chest x-rays taken in 1946 by the Gage Institute, 0.91% disclosed tuberculous disease (0.11% active) as confirmed by clinic examination; 0.61% showed evidence of significant non-tuberculous diseases or abnormalities; 1.9% showed less significant abnormalities such as previous pleurisy or pulmonary calcifications, etc.; 96.4% showed no demonstrable disease.

5. Many questions remain as yet unanswered (*e.g.*, advisability of pre-screening with tuberculin tests; how to reach more young and middle-aged adults with this service; etc.). Greater emphasis will be placed upon industrial chest x-ray surveys and "pre-employment" chest x-rays. Routine screening of hospital admissions, hospital out-patients, food handlers, etc., should be arranged. The future may require changes in strategy, but as long as this method continues to disclose a significant number of persons with asymptomatic active and inactive pulmonary tuberculosis, its adoption with the examination of contacts so revealed will constitute a useful addition to the already existing facilities for tuberculosis control.

For brevity, a section dealing with cost and an additional table showing all detailed findings, including non-tuberculous diseases according to age groups and sex, have been omitted. Such information may be obtained from the authors upon request.

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CASE REPORTS

CO-EXISTING HYPERNEPHROMA AND TUBERCULOSIS

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The co-existence of tuberculosis and carcinoma in one organ is rare, and less frequent in the kidney than in other parts of the body. The first report in the literature of such an association in the kidney seems to be that of Chomel in 1814. The next reference is found in Rayer's "Traité des Maladies des Reins" published in 1841, in which he said that in a small number of cases he had found in the same kidney "la dégéneresence tuberculeuse associé au cancer". Rosenstein in his "Pathologie und Therapie de Nieren Krankheiten" published in 1870 said he had seen only one case. None of the cases mentioned by these authors was proved microscopically, so they cannot be accepted as definitely established diagnoses.

The first scientifically authenticated case is that published by Albarran and Imbert in 1903. Since then similar cases have been reported by the following authors: Albrecht, 1905; Kyriasopoulous, 1906; Moschowitz, 1910 (papillary tumour of pelvis); Lubarsch, 1925; Braasch, Hunt, and Cooper, 1929; Lorenzetti, 1930; Olsen, 1930; Clar, 1931; Ljunggren, 1930;

and a second case in 1932. This last case was the only one up to that time in which pyelograms had been made. In 1936 Atapin reported the first case to appear in the Russian literature. He mentions the fact that Fedorob, the great Russian urologist, in his large experience had not encountered a single case. Later cases were reported by Cooper, 1932; Rupel, 1934; Keen, 1934; DeLange, 1936; Chevassu in discussing DeLange's case, mentions one of his own; Pitrolffy-Szabo, 1935; Chivalla, 1936; Alshteyn, 1936, a case of renal carbuncle associated with tuberculosis and tumour; Pepe, 1938, a case of renal sarcoma and tuberculosis; Suzuki, 1941, reported a similar case. The latter are apparently the only two cases of this type that have been reported.

Thus, as far as could be ascertained from a review of the literature until the present, only 21 microscopically proved cases of associated renal malignancy and tuberculosis have been published.

Miss E.B., an unmarried woman of 52, presented herself in May, 1939, because of gross hæmaturia which had first occurred 18 months previously and had reappeared on four separate occasions. With the last two attacks there had been experienced very severe right-sided, abdominal pain, typical of renal colic. Careful enquiry could elicit absolutely no other complaints.

The patient's past history was negative, except for a story of some suppurative process in adolescence which had required incision and drainage through openings made near the right anterior superior iliac spine. Pus is said to have drained from the wounds for a period of months, and since then there has been definite limitation of movement in the right hip joint.

Examination.—Lungs clear to auscultation and percussion. Heart slightly enlarged. Blood pressure 180/120. Tenderness in the right costo-vertebral angle. Gross scoliosis of spine and ankylosis of right hip.

Cystoscopy.—The bladder capacity was within normal limits. The mucosa was found to be studded with a number of small, yellowish areas, 1 to 2 mm. in diameter, many surrounded by a narrow zone of hyperæmia. Gross blood could be seen emerging from the right ureteral orifice.

The urine specimen obtained from the left kidney was grossly clear and negative microscopically. It yielded no growth on culture. The right specimen was bloody and contained numerous Gram-negative bacilli and many pus cells. Because of the appearance of the bladder, which suggested early tuberculous ulceration, Ziehl-Neilsen stains were done on both kidney and bladder specimens, but no acid-fast organisms were found.

X-ray examination of the abdomen with the ureteral catheters in place revealed a left kidney of normal appearance. The right kidney was at a higher level than the left and its lower pole was seen to be grossly enlarged. Upward displacement of the right kidney above the level of the left is nearly always a significant finding and when associated with a change in the contour of the organ, primary tumour must be suspected. Ljunggren in his *Studien uber Klinik und Prognose de Growitzehen Nientumoren* lays particular stress on these findings in the diagnosis of intrinsic renal neoplasm.

The left pyelogram was normal. The right was grossly abnormal and suggested two disease processes. The superior major calyx was grossly enlarged and the superior minor calyces were dilated and blunted. These findings are characteristic of inflammatory disease. The lower calyces showed the typical compression and distortion associated with renal tumour. The pyelographic findings in association with the proved infection of the right kidney led to a preoperative diagnosis of kidney tumour and pyelonephritis. Two disease entities in the same organ are not common and the association of malignancy and infection in the same kidney is unusual. From this viewpoint the case was of particular preoperative interest.

X-ray examination of the lungs was negative for metastases. The films, however, did reveal quiescent tuberculous foci in both apices and small areas of calcification elsewhere in the lung fields. The only other

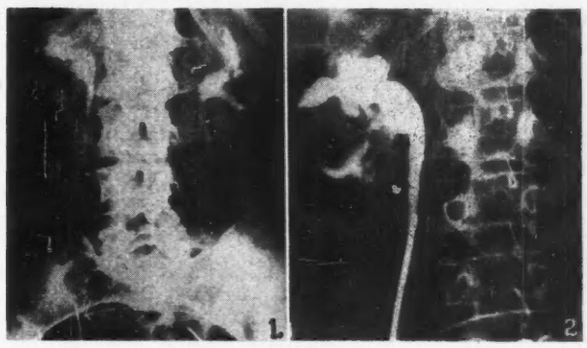


Fig. 1.—Normal left kidney. Right renal pelvis and calyces grossly distorted. Upper major calyx enlarged and minor calyces bluntly dilated. Middle calyx failed to fill. Lower calyx only partly visualized due to apparent compression. **Fig. 2.**—Right kidney reinjected showing typical deformity of intrinsic tumour. Notice expansion of lower pole.

significant laboratory findings were "marked rotoscoliosis of the spine and ankylosis of the right hip joint". This from its x-ray appearance, "suggests a quiescent inflammatory lesion, probably tuberculous".

A right nephrectomy was done. The following report on the excised specimen was received:

Macroscopic examination.—The kidney weighs 122 grams and measures 7.5 x 6.5 x 5.0 cm. The capsule is intact and some perirenal fat is attached to it. The lower pole is broadened, thickened, and bulbous. On section, a circumscribed tumour, measuring 3.5 cm. in its greatest diameter, is found occupying most of the lower pole. The lower major calyx is distorted into a slit-like crescentic space and contains a small amount of blood. The upper calyx is broadened and shortened and it also seems to be blood-stained. The periphery of the tumour lies immediately under the kidney capsule but the capsule is intact and apparently has not been involved. The tumour is distinctly yellow in colour. Its central part shows some areas of softening, small cyst-like spaces, and small foci of hæmorrhage. Grossly it appears to be a typical hypernephroma. The remainder of the kidney shows considerable parenchymatous atrophy, some increased sub-pelvic fat, and occasional small cortical cysts.

Microscopic examination.—The section taken through the tumour mass shows that it is made up of alveoli of foamy, granular polyhedral cells simulating those of the adrenal cortex. The alveoli are separated by very delicate strands of fibrous tissue. Some large thin-walled veins are present in the tumour growth and areas of both recent and old hæmorrhage. The tumour tends to be circumscribed and is pushing the renal tissue aside rather than infiltrating it. In the region of the lower major calyx the tumour contains fresh hæmorrhage. The portion of renal parenchyma lying adjacent to the tumour shows considerable scarring obstruction of

tubules and obliteration of glomeruli. No tuberculosis is found in this portion of the kidney.

A section selected from the upper pole of the kidney presents a different microscopic picture. Here in addition to considerable scarring of the renal tissue, a tuberculosis process is also present. This is manifesting itself in the form of discrete and conglomerate tubercles, some of which show central caseation. The tuberculous process appears to be active, and many of the tubercles are of recent development. The upper major calyx contains some blood and masses of purulent exudate. The pelvic lining shows small areas of ulceration, and at certain points the tuberculous process lies very close to this membrane.

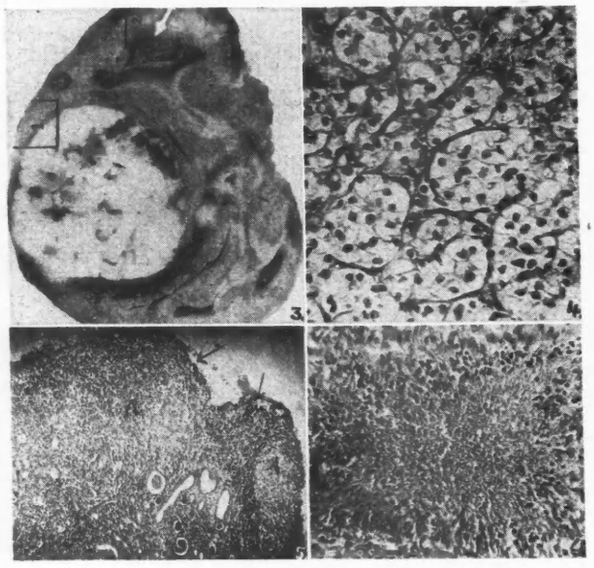


Fig. 3.—Cut surface of kidney. The paler area is the tumour mass. The white arrow indicates a tuberculous area ulcerating the pelvis of a calyx. **Fig. 4.**—High power view of tumour area, showing solid alveolar groupings of tumour cells with abundant pale foamy cytoplasm separated by trabeculæ of fibrous tissue. x460. **Fig. 5.**—Photomicrograph of area 2 outlined in Fig. 3, showing tubercle (A) and ulceration of pelvic epithelium (arrow). x60. **Fig. 6.**—A typical tubercle with caseous centre and peripheral zone of epithelioid cells. x276.

Pathological diagnosis.—Hypernephroma and tuberculosis.

The patient's recovery from the operation was uneventful. The wound healed kindly, and despite the early tuberculous lesions which had been seen preoperatively in the bladder, no vesical symptoms have appeared at any time. More than seven years have now elapsed since the kidney was removed and the patient is enjoying excellent health. Repeated urine cultures have been negative for tubercle, and a recent x-ray examination of the lungs showed no evidence of metastases.

DISCUSSION

In reviewing the symptomatology of the various cases reported in the literature it is noted that in some the symptoms of tumour have predominated as in Albrehet's, Clar's, and Lubarasch's reports. In others, notably Lorenzetti's, Olsen's, and Ljunggren's second case the tuberculous process was dominant. In the case here reported the symptoms of tumour prevailed.

No tubercles were found in the tumour itself. This was true also of some of the cases previously reported, but in others actual tuberculous involvement of the tumour or of its capsule was demonstrated.

The possible chain of events in this patient is interesting. It seems likely that she had a tuberculous arthritis in youth, although no laboratory data are available to substantiate this hypothesis. How long the kidney may have been infected cannot be determined. The tuberculous process was active at the time of operation and one can only speculate as to whether its activity was incited by the development of the tumour. Such a stimulation is occasionally seen in lymph glands which have become the seat of metastatic carcinoma.

The actual relationship between carcinoma and tuberculosis as occurring in the same individual or in the same organ has been a subject of controversy for more than a century. The earlier writers notably Walsh in 1840, and Rokitansky in 1854, claimed an antagonism existed between the two conditions. Opposition to this supposition quickly appeared, and in 1866 Van Heide described tuberculosis and carcinoma as being manifestations of the same constitutional affliction. McConkey in 1908 thought that the bacillus of tuberculosis might be the cause of carcinoma.

In more recent years a number of statistical studies have appeared. Pearl, in reviewing 7,500 autopsies at Johns' Hopkins Hospital, found 816 cases of carcinoma, 6.6% of which also had active tuberculosis. A comparable control series of 816 cases without carcinoma showed a 16.3% incidence of active tuberculosis. From this marked difference of incidence, 6.6% as compared with 16.3% in cancerous, as opposed to non-cancerous subjects, Pearl concluded that some antagonism must exist between the two conditions. Centanni and Rozzi in 1926 offered further proof of this contention. They showed that a mixture of tumour cells and bacilli injected into mice, resistant to tuberculosis, produced no growth, whereas, injections of tumour cells alone produced a growth

in most animals. They further demonstrated that dead tubercle bacilli had no inhibitory effect, whereas tuberculin did. A few years later, however, in 1929, Cherry increased the incidence of spontaneous cancer in mice by about ten times through subcutaneous injection of small numbers of tubercle bacilli.

Shortly after Pearl's publication appeared, a larger series of autopsies, 11,195, was published by Carlson and Bell, who concluded that Pearl's deductions were not justified. In support of this Fortune at about the same time studied two series of corresponding autopsy cases; in one group death was due to malignancy; in the other no malignancy was present. The incidence of tuberculosis was approximately the same in the two groups.

Thibaudeau from a study of 66,711 tissue specimens concluded that the occurrence, even though rare, of the two lesions in the same patient, often in close relationship, is against the theory of antagonism. He further indicated that the processes also occur in different age groups, and this more than anything else may make their co-existence a rarity. Oertel was apparently of the same opinion, for he said that both lesions might co-exist without marked influence on each other.

SUMMARY

A case of co-existing renal carcinoma and active tuberculosis in the same kidney is reported. Twenty-one previously reported cases are listed. The clinical, x-ray, and pathological findings are described. A brief discussion is presented of the possible relationship between the two lesions.

Thanks are due Prof. J. H. Fisher for the pathological report and the photomicrographs.

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Temperance is a bridle of gold; he who uses it rightly is more like a god than a man.

As threshing separates the corn from the chaff, so does affliction purify virtue.—Burton.

SUPERIOR LINEA SEMILUNARIS HERNIA SUBJACENT TO ARCUATE LINE*

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A white female of 31-years of age complained of a small lump in the anterior abdominal wall, about an inch and a half below the umbilicus at the lateral edge of the right rectus muscle. Coughing produced an impulse, and the mass was reducible. There was no history of direct trauma. The lump had developed over a period of two years, gradually increasing in size. Recent tenderness had made her seek medical attention. Examination also revealed a femoral hernia.

A ventral abdominal hernia was diagnosed. She was admitted for operation. The femoral hernia was easily repaired, then a vertical incision was made over the mass in the abdominal wall, conforming to the curve of the linea semilunaris. An extraperitoneal lipoma was found trailing a hernia sac, both of which had passed up under the arcuate line at the linea semilunaris, enlarging the potential triangle there, and resting subjacent to a thin anterior lamella of the internal oblique and the aponeurosis of the external oblique.

The lipoma was excised. The hernia sac containing small bowel was incised and emptied. Its base was widely dissected from adjacent tissues, to afford high ligation and to promote adhesions, and it was closed with a transfixion ligature of fine silk, then displaced in a superior-lateral position, employing the ends of the ligature for this purpose. Treatment then was simply to construct a lower arcuate line on that side. This was accomplished by cutting from the mid-line, in a medial to lateral direction, a pedicled flap about 2 cm. wide of the anterior right rectus sheath. The upper edge of the fascial strip was a little more superior than the edge of the arcuate line. The fascial flap was left attached at its lateral edge, at the linea semilunaris. It was then passed posterior to the right rectus muscle and out anteriorly through a small aperture made in the muscle near the linea alba, where its medial end was sutured to the anterior rectus sheath, from where originally it had been vertically incised.

The bare area in the rectus muscle, from where the strap had been taken, was covered by suturing the superior and inferior edges of the anterior rectus sheath, after a little dissection to remove tension in the approximation. At the lateral border of the right rectus muscle, the lax edges of the hernial orifice were sutured in an overlying fashion, with the superior edge being laid anteriorly over the inferior edge.

The patient had an uneventful recovery, and was discharged in two weeks from the hospital. During the past four years in periodical examinations, there has been no evidence of any recurrence or complication. Two years after this operation, she had an abdominal hysterectomy which gave an opportunity to examine the posterior aspect of the anterior abdominal wall. There was no weakness at any point and the region was firm and smooth with no complicating adhesions.

ANATOMICAL CONSIDERATIONS

In over 40 dissections it has been confirmed that the arcuate line is not always constant in its position. It may vary from one- to three-quarters of the distance from the umbilicus to the pubic symphysis. However, the umbilicus is a variable landmark also. The level of the anterior superior iliac spines is a more constant reference point.

* Case presented at the Herbert Reddy Memorial Hospital.

The fact that here an extra-peritoneal lipoma preceded the hernia sac suggests that such accumulations of fat may have some possible etiological significance in the production of herniæ. Lipomata have frequently been found associated with herniæ in the inguinal region. In hernia operations, they should always be carefully removed as a precaution against recurrence. An extra-peritoneal lipoma could act like an arrow-head, probing an exit with each recurring period of increased intra-abdominal pressure, and trailing a hernia sac.

Since here a case of a *superior* linea semilunaris hernia has been presented, it is assumed that it must also be possible to have an *inferior* linea semilunaris hernia. Should anyone desire a more exact anatomical terminology for a direct inguinal hernia, this may be it.

A direct inguinal hernia, going medial to the deep inferior epigastric vessels, passes out by the lateral border of the rectus muscle, the linea semilunaris, and its correct claim to the term *inguinal* has been debated before. The line of demarcation between a superior and an inferior linea semilunaris hernia would be the deep inferior epigastric vessels.

SUMMARY

A case of a superior linea semilunaris hernia is described, with a successful procedure of operative cure. The variable position of the arcuate line has been mentioned, and the possible significance of fatty tumours associated with herniæ has been pointed out. A more exact terminology for direct inguinal herniæ has been suggested.

Appreciation is extended to Professor C. P. Martin, M.A., M.B., Sc.D.,(Dublin), Robert Reford Professor of Anatomy, and Chairman of the Department of Anatomy, Histology and Embryology of McGill University, for reviewing this paper before publication.

There have been admirers who claimed for Pasteur more than he had done. That is unnecessary and does not help his memory. He did in his lifetime sufficient to make ten men great. That is enough. It is a pity to gild the lily. He was one of those rare individuals thrown up at intervals throughout the world's history to restore order out of chaos. He was a great Frenchman, but it is not only France which has to thank him. The whole world is deeply in his debt.—Sir Alexander Fleming, *Brit. M. J.*, April 19, 1947.

FATAL CASE OF TOXIC JAUNDICE DUE TO T.N.T.

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The signs and symptoms of T.N.T. sickness have been discussed in another paper in this issue. The case reported below illustrates most of the features of severe T.N.T. sickness and includes an autopsy report.

This patient was a young man of 23 years. He worked in T.N.T. from January 4 to February 1, 1942. On January 29, he received a first and second degree burn from molten T.N.T. on the inner side of the left wrist approximately 1½ inches in diameter.

He was treated within half an hour and the burn was dressed daily for three days and he continued to work in T.N.T. until February 2, when he changed to the paint shop and did not return to T.N.T. The burn healed by February 8.

On February 24, he reported to the First Aid nurse with epigastric pain and nausea. She noticed that he had jaundice and referred him at once to the plant hospital, where he was given a stomach mixture and allowed to remain at work.

On February 27 he was seen again at the hospital out-patient clinic. It was noted that his jaundice was becoming more intense and he was admitted to hospital. He was placed on a fat-free, high carbohydrate diet with abundant fluids and was given 2 drams of a saturated solution of magnesium sulphate every morning.

Urine examination on February 27; Webster test negative; specific gravity 1.020; acid, no albumen; no sugar; some bile. White blood cells 5,000; neutrophils 54%; eosinophils 1%; basophils 0; lymphocytes 36%; monocytes 9%; non-filamented neutrophils 11%. The white blood cells appeared normal in the smear. Red blood cells 5,000,000; Hgb. 99% (15.4 gm.); hæmatocrit 45 c.c. of packed cells per 100 c.c. of blood; volume index 0.99; colour index 0.98; saturation index 0.99; reticulocyte count 0.5%; icterus index 50 units. The red cells were normal in appearance and appeared to be well filled with hæmoglobin.

On March 1, the bleeding time was found to be 1½ minutes; coagulation time 6 minutes; clot retraction time 1½ hours; capillary resistance test negative.

Throughout his stay in hospital from February 27, until March 7, 1942, the fact that he had been previously exposed to T.N.T. was unknown to his medical attendants. He was asked if he had ever worked in T.N.T. and he said he had not and it was taken for granted that he had been working only in the paint shop where he was employed at the time of his admission to hospital. The clinical record of his T.N.T. burn was not discovered until March 21.

Being unable to obtain from the patient a history of exposure to T.N.T. the jaundice was supposed to be non-occupational. A provisional diagnosis of obstructive jaundice was made. He made some improvement while in hospital having an increase in appetite. Bile pigment was present in the stools from time to time.

While in hospital, his chief complaints were weakness, fatigue, loss of appetite, and nausea. He did not complain of itchiness of the skin. Nothing was found on examination except the marked jaundice. There were no abnormal masses found in the abdomen and no tenderness in the vicinity of the liver and bile ducts. The stools were clay coloured most of the time. His temperature remained normal.

* Director Health Division Atomic Energy Project, National Research Council.

On March 7, he was somewhat improved and was discharged from the plant hospital. Little is known of what he did after his discharge from hospital but it seems he spent very little of his time at home. He appeared again on March 19, at the out-patient clinic where he was seen by me. He was obviously much worse, more deeply jaundiced, weak and ill. His heart rate was 96.

He was re-admitted to hospital on March 19 and was again given a low fat, high carbohydrate diet and kept in bed. He became progressively worse with intense jaundice, weakness and apathy and his heart became irregular.

On March 21, his accident card covering his T.N.T. burn was discovered and his exposure to T.N.T. established. We then obtained the history of his work in T.N.T. He said that while working in T.N.T. he washed his hands and face when he went off work every time. He skipped the required daily shower bath after work but showered about twice a week. He changed his overalls and cap twice a week. He wore the same underwear off work as when at work but changed it regularly once a week. He put on clean socks about every two days.

While in hospital on this occasion his temperature reached as high as 99.8° F.

Blood examination on March 21, 1942, was as follows: white blood cells 5,000; neutrophils 47%; eosinophils 1%; basophils 4%; lymphocytes 41%; monocytes 7%; nonfilamented neutrophils 7%. Red blood cells 4,500,000; Hgb. 97%; reticulocyte count 0; icterus index, over 100 units.

On March 21, the patient was transferred to the Toronto General Hospital where it was noted that his liver appeared to be reduced in size and the spleen not palpable. The urine was found to contain a trace of albumen, bile four plus, and numerous granular casts.

The van den Bergh was 53 units, carbon dioxide combining power was normal. He was given 10% glucose intravenously but became rapidly worse and died on March 23, 1942.

Autopsy report (Dr. I. H. Erb).—The autopsy was performed 3 days after death and after the body had been embalmed. Many of the organs had been punctured by the embalmer's trochar. The significant findings were as follows:

The pancreas and adrenals showed some post mortem change but no significant lesions. There was early bronchopneumonia of the right lower lobe with some overlying acute pleurisy. The conjunctivæ, skin, pericardium, pleura, lymph glands, were deeply jaundiced. There was slight jaundice of the inner and outer surfaces of the dura mater and the cerebrospinal fluid in the cerebral ventricles was of a yellowish tint. The heart which weighed 380 grams, was punctured in many places and the blood had been removed. The various chambers and valves all appeared healthy except that the endocardium and myocardium both had a greenish-yellow tint. The coronary arteries were healthy but there was some early atheroma of the ascending portion of the aortic arch.

The liver was small, weighing 1,195 grams. Its outer surface was uneven and distinctly mottled. It showed many punctures. The general background was brownish red, but standing out in marked contrast were numerous dark greenish-yellow, slightly elevated, irregular outlined patches of liver, varying from a few mm. up to 5 cm. in diameter. It was fairly firm but had been embalmed. The cut surface showed the same raised mottling but with still more contrast. The green areas again tended to be elevated and comprised approximately one-third of the liver substance. These green areas also presented a fine mottling, small very dark green areas, separated by pale green elongated irregular bars of tissue.

The gall bladder was punctured. It contained about 20 c.c. of very dark green thick bile. The mucosa appeared healthy, except for the deep green staining of its surface.

Kidneys and Ureters.—Both kidneys were somewhat enlarged, the right weighing 230 grams and the left 250 grams. Both appeared greenish through the capsule. On section the edges everted slightly (this might have been more marked but for the embalming). The cut surfaces of the cortex and medulla presented a rather marked contrast, the former being quite green with slight fine reddish mottling, while the medulla presented a pale red background in which numerous dark red streaks radiated outward to the bases of the pyramids, while the apices were of a pale greyish red colour. The cortex measured 5 to 15 mm. in thickness, the medulla 10 to 25 mm. The pelves were stained slightly green. Ureters healthy.

Anatomical diagnosis.—(1) Subacute yellow atrophy of the liver. (2) General jaundice. (3) Subacute nephrosis. (4) Bronchopneumonia, right lower lobe. (5) Acute pleurisy, right lower lobe. (6) Early atheroma of aorta. (7) Multiple embalmer's wounds.

Microscopic examination.—The microscopic findings of interest were confined to the liver and kidneys.

Liver: The general structure of the organ was greatly altered. In the raised green areas in the gross, the liver lobules were still recognizable, but the central and midzonal areas showed marked disorganization of structure and degeneration of cells, many of which remained as deeply pigmented, non-nucleated "ghost" cells, varying greatly in size and contour. Around the periphery of some of the lobules a zone of fairly well preserved liver cells could be seen. The bile capillaries adjacent to these better preserved areas appeared relatively normal, but there was some lymphocytic infiltration in the periportal tissues.

In the areas which appeared red and depressed in the gross (approximately two-thirds of the liver substance) no parenchymatous cells remained. The central veins stood out prominently and these were surrounded by the collapsed connective tissue framework of the lobule in which could be seen small granules of brownish pigment and indefinite capillaries containing red blood cells. In the original periportal areas there was considerable lymphocytic infiltration and here there was already quite marked proliferation of bile capillaries but no evidence of new liver cells.

Sections stained with scarlet red were negative for fat.

Kidney: The glomeruli appeared healthy except for slight post mortem change. The cells of the proximal convoluted tubules showed marked degeneration although still pretty well maintaining their shape; the nuclei no longer stained (this was probably largely post mortem degeneration). The collecting tubules were better preserved. In many of the tubules could be seen greenish acellular casts. The capillaries of the medulla were deeply congested.

Bone marrow: Sections were studied from sternum and vertebræ. Both presented essentially the same picture. The bone marrow was cellular and appeared active. Nucleated reds could be seen; also numerous cells of the granular series, both neutrophile and eosinophile.

Summary.—The immediate cause of death in the pathologist's opinion was hæmorrhagic bronchopneumonia complicating severe liver and kidney damage. The picture in general was that of acute yellow atrophy of the liver which had progressed to the stage in which much of the liver parenchyma had disappeared and in which repair was beginning as shown by the proliferation of bile capillaries. The damage was so extensive as to lead to the belief that had pneumonia not intervened, the outcome would still have been fatal. Given a history of exposure to T.N.T. it is believed that this was the etiological agent.

There are several important points in this case which might be reviewed here.

1. The time of onset of the toxic jaundice is unknown. No jaundice was noted while he was

under treatment for the T.N.T. burn from January 29 to February 7. Jaundice was, however, very marked when he was seen next on February 24.

2. Exposure to T.N.T. was repeatedly denied by the patient which confused the diagnosis until it was too late to render proper treatment.

3. The importance of the burned area as an avenue of absorption of T.N.T. is uncertain because no notice of constitutional signs and symptoms was taken at the time the burn was under treatment.

4. This case of toxic jaundice was encountered early in the operation of the plant at a time when knowledge and experience with T.N.T. poisoning was very limited.

5. This case emphasizes the fact that younger people are more susceptible to T.N.T. poisoning. It further brings home to the clinician the importance of early diagnosis and early and adequate treatment.

A CASE OF CAVERNOUS SINUS THROMBOSIS WITH RECOVERY*

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Cavernous sinus thrombosis is an uncommon intracranial infection from which until recently recovery was rare. The best treatment known at the present time appears to be large doses of penicillin and of sulfadiazine. However the therapy of cavernous sinus thrombosis cannot be standardized as only about 15 cases treated with penicillin have been recorded in the medical literature of English-speaking countries. This number is insufficient to do more than suggest what should be done and emphasizes the need to record further experience. Hence the following case, treated with penicillin and sulfadiazine, is being reported.

Thrombophlebitis of the cavernous sinus was first described in 1821, and recovery of a patient with this condition following a surgical procedure was first reported in 1893. Since then attempts have been made to drain the cavernous sinus through the orbit, through the nose and sphenoidal sinus, and along the middle cranial fossa. These radical measures were sometimes successful. More simple operations have been ligation of the angular vein in cases of infections of the face, and ligation of the internal carotid artery to keep the cavernous sinus at rest. Medical treatment has included the use of antitoxin, bacteriophage, and more recently, heparin, sulfonamides and penicillin. Even before the days of sulfonamides and penicillin recovery of cases of cavernous sinus thrombosis was known to occur but the mortality was high. Grove[1] in 1936 estimated the mortality to be over 93%. A list of recoveries collected from all the literature and published in 1943 by MacNeal *et al.*,[2] contains only 58 cases. In a series of 45 cases treated with bacteriophage between 1931 and 1940 there was a mortality of 69%.[3]

Lately, cases have been reported in which sulfonamides and penicillin were used with satisfactory results. In 1944 Wolf[4] reported recovery in a case of cavernous sinus thrombosis following use of 64 gm. of sulfadiazine and 500,000 units of penicillin. A total of 2,000,000 units of penicillin with sulfadiazine and heparin were used with success in 1944 by Wiesenfeld and Phillips[5] in another case. Orbital cellulitis was suspected and the orbit was explored in a case of thrombophlebitis of the cavernous sinus described by Welty[6] in 1946. In this case a total of 3,600,000 units of penicillin as well as sulfadiazine and heparin were given. Sears and Wilson[7] describe a case of staphylococcal pneumonia complicated by cavernous sinus thrombosis in which the organism was only partially inhibited by penicillin. A total of 13,200,000 units of penicillin was given, together with 150 gm. of sulfadiazine and specific antiserum. The patient recovered.

Not in every case is recovery complete, even though life is saved. Ocular paresis and blindness, partial or complete, are not infrequent sequelæ.[6, 8, 9] Prompt diagnosis and early intensive therapy with penicillin and sulfadiazine appear to be the best way to effect recovery as well as to safeguard against permanent disability. The tendency has been to give penicillin in doses of 10,000 to 30,000 units every three hours up to a total of about 2,000,000 units.[5, 10, 11, 12] In the case reported by Belsky[13] (1945) penicillin was increased progressively from 25,000 units to 100,000 units every four hours because there was no response with the lesser dosages. A total of 6,000,000 units was given with a favourable result.

Small doses of penicillin for a short time are not likely to produce satisfactory results. Wolfe and Gain[14] (1945) report a case of thrombosis of the cavernous sinus where the organism was resistant to sulfonamides but was sensitive to penicillin. There was steady improvement with an average intramuscular dose of 10,000 units every three hours for 9 days, after which penicillin was discontinued. About three weeks later the patient developed signs

* From the Service of Dr. I. J. Patton, Homœopathic Hospital of Montreal.

of a brain abscess. He was given penicillin 10,000 units every three hours again but this was of no avail. There were abscesses in the brain and lungs at autopsy.

M.G., a poorly nourished but otherwise healthy girl of 16, was seen in the out-patient clinic of the Homœopathic Hospital of Montreal on May 14, 1947, with a furuncle on the left side of the face which had been present for five days. She had received about 600,000 units of penicillin in oil and sulfonamides from an outside doctor in the previous two days. When she came in she had a fever of 101.5° F. and there was a little cellulitis around the boil which seemed well localized over the left zygoma. The core of the boil appeared to be free and was very carefully lifted out. She was sent home, told to rest, to apply hot compresses, to drink fluids freely, and to return the following day. She was not given sulfonamides as apparently she had not tolerated them well. The following day the patient was brought in by ambulance, semi-conscious and delirious.

On admission (7 p.m., May 15) her temperature was 105, pulse 108, respirations 32. At this time there was a small opening at the centre of the boil with gross inflammatory swelling and induration extending to the temple and parotid region. There was ptosis of the left eyelid, the left pupil was smaller than the right, but both reacted to light. There was some stiffness of the neck. The deep reflexes were equally active and the plantar responses were flexor. The heart and lungs were free of abnormal findings. The cerebrospinal fluid showed a pressure of 200 mm. and was clear except for a few fibrinous shreds. Numerous polymorphonuclear cells and lymphocytes were seen, and what were thought to be intracellular Gram-positive diplococci were reported in large numbers. The fluid was cultured but no growth was obtained. No organisms were seen in later samples of spinal fluid and as the original slides were lost the presence of intracellular diplococci could not be confirmed. A blood culture was taken but no growth obtained.

The diagnostic impression was staphylococcal facial cellulitis with cavernous sinus thrombosis and beginning meningitis. Treatment was instituted with penicillin 75,000 units every three hours intramuscularly and sulfadiazine 2.5 gm. intravenously together with 1,000 c.c. of 5% glucose in normal saline. The sulfadiazine and fluids were repeated twice during the first night in the same quantities. Penicillin 30,000 units in 10 c.c. normal saline was injected intrathecally during the night.

The next morning, May 16, the patient was restless, irritable, and somewhat delirious. Swelling of the face had increased and included the left eye, temple, cheek and side of the neck. There was slight proptosis of the left eye as well as ptosis and miosis; upward, inward and downward movement of the left eye was limited. The fundi appeared normal. Definite trismus was present, and a swelling of the left side of the throat which could not be clearly seen. The possibility of orbital cellulitis was considered but cavernous sinus thrombosis seemed to be the more likely diagnosis.

Two intrathecal injections of penicillin of 30,000 and 50,000 units in 10 c.c. saline were given during the day. As the patient was not improving in the evening the intramuscular dose of penicillin was increased to 150,000 units every three hours.

From May 17, improvement began and continued uneventfully. The swelling of the face subsided and the ocular signs gradually disappeared except for a paresis of the left external rectus and poor co-ordination of the left eye with the right. The patient was discharged on June 3. She was seen again on June 24 in the out-patient clinic and then showed much better movement of the left eye with only a minimal paresis of the left external rectus. Some lack of co-ordination of the left eye was still present. In August the eye movements were better co-ordinated and there was still some residual paresis of the left external rectus.

DISCUSSION

Treatment.—The causative organism in this case was assumed to be *Staph. pyogenes* and the treatment was based on this assumption. With the severity of the infection it was felt that intensive therapy with penicillin and sulfonamides was needed. Penicillin 75,000 units every three hours intramuscularly and 30,000 units twice a day intrathecally were given, together with sulfadiazine because it would penetrate the meninges. After 24 hours with no improvement the penicillin was raised to 150,000 units every three hours until the fifth day when there was sufficient improvement to warrant lowering the dose to 75,000 units again. It was deemed unnecessary to continue intrathecal administration of penicillin after three doses. Penicillin was gradually lowered to 25,000 units every three hours and was continued at this level until the day before discharge. A total of 9,475,000 units was given in a period of 18 days. Sulfadiazine 1.0 gm. every four hours was given for 11 days. Satisfactory blood and spinal fluid levels were obtained with this dosage and leucocyte counts and urinalyses showed no toxic effects from the prolonged use of this drug. A persistent headache, which stopped on the day after sulfadiazine was discontinued, may possibly have been due to the drug. The total amount of sulfadiazine given was 70 gm.

It is felt that the dose of 150,000 units of penicillin every three hours was justified by virtue of the serious nature of the illness and because, theoretically at least, one is attempting to penetrate the depths of a septic thrombus. Therapy should be continued until all clinical evidence of activity has been absent for about one week.[15] On the other hand it is felt that the amount of penicillin given intrathecally was dangerously high. Walker *et al.*[16] (1946) report that 40,000 units of penicillin intrathecally may produce generalized convulsions followed in some cases by coma and death. These undesirable effects occurred with ten different makes of penicillin and also with purified crystalline penicillin and are therefore not attributed to toxic impurities. A more suitable dose of penicillin would have been 10,000 to 20,000 units once or twice a day.[17]

The use of heparin and staphylococcal antitoxin was considered but neither of these preparations was given. Heparin has frequently been used in the treatment of cavernous sinus thrombosis.[5, 6, 13, 18 to 21] By increasing the blood clotting time it tends to prevent further formation of thrombus. But the value of heparin in the treatment of this condition has not been established[22] and such complications as cerebral petechial hæmorrhages and massive hæmaturia[23] have been known to occur. If heparin is used it must be given with great care and frequent adjustment of the dose to the clotting time desired (about 15 minutes). It seems, however, best not to use it because, at the present time, it appears to be an unessential and potentially dangerous adjunct.

DIAGNOSIS

Eagleton[24] specifies six points on which rest the diagnosis of cavernous sinus thrombosis. According to him, there must be: (1) a known site of infection; (2) blood stream infection; (3) venous obstruction (shown by fullness of retinal veins, proptosis or papillœdema); (4) nerve involvement (cranial III, IV, V and VI); (5) abscesses in adjacent soft parts (such as brain, pterygoid plexus); (6) complications such as pulmonary infarcts, meningitis. As early diagnosis and intensive therapy are imperative for success in treatment one is justified in not awaiting the appearance of all these diagnostic criteria. In cases recently reported seldom are all these points demonstrated.

Infections of the face are probably the most common source of cavernous sinus thrombosis, but the site of infection may be the ear, the paranasal sinuses, a tonsil or even a tooth. Interference with an infection on the face is notorious for its effect in precipitating cavernous sinus thrombosis and it is possible that in the present case lifting out the core of the boil helped to spread the infection.

Bacteriological evidence of blood stream infection is usually readily obtained, but it was missing in the present case probably because of previous administration of penicillin. Nor was there any fullness of the retinal veins; but the proptosis most probably was caused by venous obstruction. There was good evidence of involvement of the III, IV and VI cranial nerves, and the presumptive diagnosis of cavernous sinus thrombosis was based on the ocular palsies, ptosis, proptosis and signs of meningeal irritation. At the outset a secondary peritonsillar abscess was suspected but trismus prevented accurate inspection of the throat. The trismus could have been due to the proximity of the primary infection to the temporalis and masseter muscles. As the swelling of the face subsided the trismus disappeared and no throat infection was seen.

The question arises whether orbital cellulitis alone could have presented the symptoms and signs found in this case. In orbital cellulitis a more marked local reaction with chemosis, œdema of the eyelid and severe pain in the orbit would be expected. The fact that the VI cranial nerve was still involved after other ocular signs had disappeared leads one to think that the pathological process had originally extended to the cavernous sinus where the abducent nerve is relatively more exposed than the other cranial nerves which pass through this space. The delirium, stiff neck and cerebro-spinal fluid findings are a further indication that the disease was not confined to the orbit.

SUMMARY

1. Until the advent of sulfonamides and penicillin cavernous sinus thrombosis was a disease with a mortality between 69 and 93%.

2. The present mortality figures are undoubtedly much lower but are not known because of the few cases reported. Only about 15 cases of cavernous sinus thrombosis treated with penicillin and sulfonamides are to be found in the medical literature of English-speaking countries.

3. Early intensive therapy with penicillin and sulfadiazine is suggested as the best treatment. Penicillin should be given in dosages as high as 150,000 units every three hours and 15,000 units twice a day intrathecally, to be decreased with clinical improvement. Treatment should be continued for one week after clinical signs of activity have disappeared. Heparin appears to be unessential and potentially dangerous.

4. A case of cavernous sinus thrombosis is described in which the treatment outlined was used with a satisfactory result.

Grateful acknowledgment is made to Dr. F. L. McNaughton for assistance in the preparation of this paper.

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TRANSECTION OF ILEUM BY A BAND

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It is desired to report this case history for two reasons: firstly that through an error of judgment the patient perished, secondly that a band can be an anvil against which successive waves of peristalsis hammer the ileum until it severs itself utterly and completely.

The patient was a girl aged 8. Apart from measles in infancy she had been well until December, 1946, when an acutely inflamed appendix had been removed via a right rectus incision. There had been some slight wound sepsis. She was discharged on the eighth postoperative day, remaining well until eight days before she was seen by me in consultation. She had been eight days in hospital with the following story. On April 14, 1947, she suddenly developed central abdominal pains which were colicky. She vomited several times with the initial pains, but after that had no further vomitings. Her general condition had slowly deteriorated. The white cell count at the onset was 11,000. The urine showed acetone only and there were no microscopic abnormalities. The abdomen slowly became distended, showing peristaltic waves. A plain skiagram of the belly showed coils of ileum leading down to the right lower quadrant. A barium meal had been given and confirmed the above "flat plate". Two enemas produced fæces in each specimen.

Examination; 2400 hours April 22, 1947; the little girl was tall for her age, and slim. She looked ill, but not toxic. The tongue was clean and moist, the pulse 102. Examination of head, neck, thorax and nervous system showed no abnormality.

The belly was greatly distended, but not tender. There were visible coils of bowel forming a ladder pattern but peristaltic waves were not visible, though one waited and watched for them. Auscultation elicited loud coarse obstructive noises and fine tinkling gurgling fluid-and-gas sounds. Bimanually, with one finger in the rectum, it was obvious that there was much more material in the right lower quadrant than in the left lower quadrant.

It was not difficult to diagnose a late low mechanical obstruction of the ileum, most likely by a band, whose source was the previous laparotomy. Arrangements were therefore made for continuous gastric suction via a Ryle's tube, which was passed via the nose, and saline with 2½% glucose solution was given intravenously by drip-control. At this point I was persuaded that the patient would improve temporarily on the above regimen and "be fitter for operation in the morning", since she had already been ill for eight days.

At 0400 hours on April 23, 1947, the patient suddenly developed severe abdominal pain. She did not vomit. Examination showed a collapsed magenta-hued patient. Pulse 112 and thready. There was much less abdominal distension than when previously seen; and the suction bottles had been very effective. The blood pressure was 68/50. The belly wall was board-like, and signs of free fluid and gas were present; *viz.*, hyperresonance centrally, dull flanks, and silence on auscultation.

Operation.—Cyclopropane and oxygen for 58 minutes. Through a right paramedian (para-central) incision free gas, barium solution and small bowel contents were seen and sucked away. Two bands were found in the right lower quadrant. They were omental bands and stretched from that structure to their points of anchorage in the right iliac fossa. One band was white, tough and avascular, just like a shoelace. It had actually cut the terminal ileum cleanly across, the proximal gut (within two hours of the crisis) being now closed by spasm, neither any bleeding having occurred at any time, nor any further contents being allowed to escape after the first enormous gush. The distal cut end was open and pouting and likewise had not bled at all; this section of ileum was of course constricted throughout, being empty and unobstructed. Both ends were rapidly closed and an antiperistaltic anastomosis carried out; the remaining peritoneal filth was sucked out and the wound in the abdomen closed. During the operation the blood pressure remained between 68/50 and 72/50. The patient died at 1000 hours on April 23, 1947 without regaining full consciousness. The usual penicillin and sulfathiazole were of course given, but without hope.

The above case notes illustrate how the ileum may be completely cut across by the forces of nature acting against an internal band, and that the ileum has the power to seal off rapidly. The lesion was a sudden and complete rupture of an annular gangrenous constriction of the terminal ileum. Further, it is folly to rely on gastric suction alone, in the presence of visible peristalsis, even when a pure mechanical lesion has been diagnosed with confidence.

Anyone who has had the experience of giving real psychiatric service, not merely consultations, in any area where it has not been used, knows that in a matter of weeks he is swamped with demands. The minute that you start to give service in this field the demands grow out of hand: and this is simply one of the basic facts of human life that we have to accept. It is the challenge that confronts us.—Lawrence S. Kubie.

CLINICAL and LABORATORY NOTES

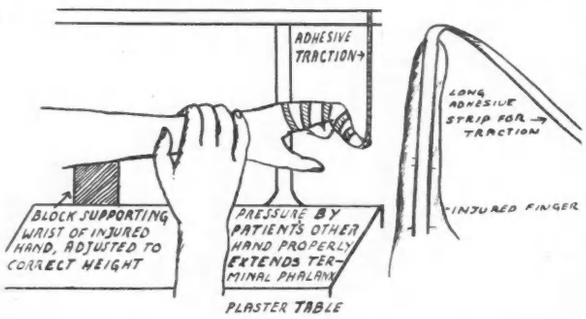
TREATMENT OF MALLET FINGER

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Frequently, particularly in the summer months, many of us are confronted with injuries described as "mallet finger" or "baseball finger". A method I have found highly successful in treating these injuries might prove of interest. Use of the conventional Lewin's (almost straight) splint of metal or plastic, with the end bent up, has the disadvantage and objection that it often slips off and it is frequently difficult to maintain adequate and full extension of the distal phalanx, a desired condition in treatment, for reasonably quick healing, best results and limitation of period of disability. Full flexion of the proximal interphalangeal joint, thereby relaxing the flexor tendon and thus aiding full extension of the distal phalanx is important.

In the method I use, a strip of adhesive or elastoplast about 4′ long and ⅜″ wide is stuck to the anterior surface of injured finger, one end extending just above the proximal phalanx into the palm, the other end carried over the distal phalanx, after the proximal interphalangeal joint has been fully flexed. A strip of adhesive about ¼″ wide is now firmly, but not tightly, wound, with enough turns to hold securely, first close to the proximal end of the proximal phalanx, and then is carried in a figure of eight fashion around the proximal



inter-phalangeal joint to the distal end of the middle phalanx where sufficient turns are taken to hold securely. The result is firmly fixed flexion of the proximal interphalangeal joint. A strip of adhesive ¼″ wide is then wound around the dorsum of the proximal interphalangeal joint, carrying adhesive as near as possible to the distal end of the middle phalanx and the proximal end of proximal phalanx, to further maintain full flexion of the proximal inter-phalangeal joint.

Stockinette, and if desired, sheet wadding are now applied to finger, and 4′ strip of adhesive fixed along the anterior surface of finger, with fixation reinforced by the described turns of adhesive around finger, is now raised and fastened to a bar 3 to 4′ above plaster table. Patient is now instructed to apply steady, firm pressure on the dorsum of the injured hand, with his other hand, thereby securing traction and full and steady extension of the distal phalanx. Pressure by patient is adjusted until the desired extension of the distal phalanx is secured and then, so steadily maintained. Plaster cast of sufficient rounds to secure adequate and firm fixation is now applied to the whole finger and when set yields firm proper extended position of the distal phalanx. Where there is swelling, cut or infection, cast is applied as mentioned, but changed in a few days as indicated, or a window can be cut in cast for dressings, etc. If it is desired the cast can easily be bi-valved, removed, and retaining adhesive pulled off, the two shells are then reapplied and fixed with bandage or adhesive. This has the additional advantage of allowing daily inspection if necessary and dressings, etc. Later one shell only can be applied, thus permitting massage, light treatment, etc.

Many of these injuries, in my experience at least, are seen a week to a month or more after injury and thus in some cases at least, end results by immobilization and extension alone, no matter how long carried out, often leave much to be desired and fall short of full recovery of function. The procedure outlined can easily be carried out in a matter of minutes and patient's satisfaction is certainly gratifying.

Results in my cases, using the above method, have been entirely satisfactory in every injury treated, even in one case of five weeks' standing. Length of time of immobilization is an individual characteristic and must be adjusted by progress of healing whether slow or fast, as found at the inspections of each injury from time to time. Removal of adhesive is not essential other than in cases allergic to adhesive. Its retention causes little or no inconvenience or discomfort if carefully applied.

This method may not be new, but a description of it has never come to my attention. With little effort, none on the part of the surgeon, the long adhesive strip maintains firm steady and satisfactory position of the terminal phalanx, while the plaster is being applied. The advantages of this procedure, among others, are the unhindered ease with which the plaster cast can be applied; the firm fixation obtained in the desired position; and the comfort of the patient, as there is little possibility of harmful movement and none of cast slipping off. An additional steadying device can be added by placing a block 10 to 12″ in height under the wrist of the injured hand, and this can easily be adjusted to the most convenient height, while the plaster cast is being applied.

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SPONTANEOUS UTERINE RUPTURE*

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THE following case is one of unusual interest:

The patient was a married woman 37 years old. She had six living children, the last one seven years ago. She was now in her seventh pregnancy which had had a normal course until five days before her admission to our hospital. At 5 o'clock in the morning of that day, the membranes ruptured and the patient was transported to a private clinic. However the labour pains stopped before any cervical dilatation occurred and nothing happened for a few days. On the afternoon of the 11th, the pains reappeared and there was a profuse bloody vaginal discharge.

At 9.00 that night the patient had a sudden sharp pain of almost syncopal intensity, located on the left side of the abdomen. It was followed by a temporary remission and a disappearance of the uterine contractions. An obstetrician was called in consultation and he diagnosed a premature separation of the placenta. He advised immediate transportation to a general hospital for an emergency Cæsarean section. While in the private clinic, the patient had not been subjected to any obstetrical manipulations nor did she receive any oxytocic substance.

When I arrived at the operation room, in response to an emergency summons the patient was already on the table. Her nose was pinched, her face was ashen, her mucous membranes were very pale. Her blood pressure was 90/40, her pulse 160 and her temperature 97° F.

The abdominal examination showed an enormous abdomen which was so tense that the patient had difficulty in breathing. There was a generalized contracture of the abdominal wall, but on careful palpation we had the impression that the fetus was free in the abdominal cavity since we could feel a limb. A transfusion had already been set up and we proceeded immediately to perform a median suprapubic laparotomy.

On opening the abdomen it was found full of blood and clots and the uterus was ruptured along its entire left side from the fundus to the isthmus. The lateral vaginal wall and the left broad ligament were also ruptured completely. The fetus and the placenta were in the abdomen. The left uterine artery had been ruptured and the proximal segment had been dissected out of the uterine wall for a length of three cm. This free portion was folded over on itself and so compressed by the weight of the fetus that there was very little bleeding from the artery. This fortunate disposition was certainly responsible for the survival of the patient up to the time of the operation; for, when we removed the fetus, the artery bled so fast that the entire operative field was covered with blood before we could clamp it.

The total time for the extraction of the dead fetus and placenta plus a sub-total hysterectomy with a bilateral annexectomy and an appendicectomy was 35 minutes. The operation was over 1 hr. and 40 min. after the admission of the patient.

During the operation 1,000 c.c. of blood were given by a high-speed transfusion and 1,000 c.c. of saline and dextrose solution. At the end of operation the blood pressure had risen to 130/90 and next morning after a second transfusion it was at 104/60. The red blood cell count was 2,660,000 and the Hb. 7.80 gm.

The postoperative course was uneventful and the patient was discharged twenty days later.

The pathological report did not give any histological reason for the rupture, which we think was due to a premature separation of the placenta with subsequent uterine distension caused by the accumulation of blood in the uterine cavity. This accumulation was due to a block caused by either a contraction of the cervix or a failure of the membranes to separate from the uterine wall with the formation of a closed cavity.

We attribute the greater part of our success in this case to the prompt operation and to the rapid transfusion.

This unusual case so interested us, that we made a brief survey of the current literature on the subject. From the statistics that we found and from ours, we drew the following conclusions:

FREQUENCY

The reported frequency varies greatly amongst the authors, but they all report a relatively high frequency. This had not been our experience.

TABLE I.—FREQUENCY

Author	*Deliveries*	*Ruptures*	*Incidence*
Sheldon	47,554	26	1: 1,829.0
Burkons	17,793	12	1: 1,482.9
Knowles	6,009	12	1: 1,829.0
Dugger	318,103	105	1: 3,029.5
Acken	25,935	15	1: 1,729.0
Davis	147,625	184	1: 802.3
Sadowsky	9,079	13	1: 698.4
Eastman	53,574	53	1: 1,010.6
Morrison and Douglass	65,916	45	1: 1,464.8
Present report (*cf.* Table III.)	45,462	4	1:11,365.0

PREDISPOSING CAUSES

The predisposing causes of the two types of rupture are many and we summed them up in the following classification. (See Table II.)

MULTIPARITY

Many authors give multiparity as one of the main predisposing causes of rupture and say; "As the number of pregnancies increase, the uterine musculature becomes definitely scarred. The myometrium becomes pathological due to fatty or hyaline degeneration of the uterine muscle." It is our opinion that multiparity by itself should be rejected as a predisposing cause; because, although it is well-known that French-Canadian women have large families, as high as 15 or 20 children, a survey of the four largest French hospitals of Montreal re-

* Read at the Seventy-eighth Annual Meeting of the Canadian Medical Association, Section of Surgery, Winnipeg, June 26, 1947.

TABLE II.—PREDISPOSING CAUSES AND CLASSIFICATION OF RUPTURES

SPONTANEOUS	Post-Cæsarean section (scar)			
	No previous Cæsarean section	Weakening of the uterine wall (degeneration)	Previous operation	suspension, curettage, etc.
			Tumours	
			Septic processes	
			Multiparity ???	
		Mechanical factors	Fetal dystocia	hydrocephalus, malpresentation, etc.
			Maternal dystocia	cervical scarring, colporrhaphy, etc.
			Misuse of oxytocic substances (pituitary extract)	

1 case ↑ (Proportion) ↓ 9 cases

TRAUMATIC	Manual removal of the placenta	at the time of Cæsarean section, artificial delivery
	Forceps	
	Internal version	

TABLE III.—INCIDENCE FOR FRENCH-CANADIAN WOMEN

Hospitals	*Period*	*Deliveries*	*Cæsarean section*	*Uterine rupture* — *Cases*	*Mortality* — *Maternal*	*Fetal*
H.G.M.	1927 to 1947	18,116	149	1	1	1
H.N.D.	1937 to 1945	12,749	194	1	1	1
Ste. J.	1935 to 1946	10,726	178	2	1	2
St. L.	1941 to 1945	3,871	31	0	0	0
Total		45,462	552	4		
			Ratio: 1:11,365			

vealed that the incidence of ruptures among them is very low, much under the average for the English hospitals of the country.

The symptoms of incomplete rupture differ totally from those of complete rupture.

In incomplete rupture, the symptoms can be minimal and so obscured as to lead us to almost inevitable diagnostic errors. Many such mistaken diagnoses may be found in the literature; for example, in one case, a uterine rupture was overlooked during a Cæsarean section and it was only three days later, while the patient was reoperated on for continuing pains and an enlarging abdominal mass, that it was recognized.

In complete rupture, the symptoms are usually dramatic. To the primary shock due to the rupture are added the signs of an important internal hæmorrhage. The contractions cease and some part of the fetus can usually be palpated. If there is sufficient cervical dilatation, the vaginal examination may reveal intestinal loops or parts of the greater omentum in the uterine cavity. There is also considerable abdominal distension with dyspnœa, caused by the compression of the thoracic cavity.

The operative findings will vary according to whether we are in presence of a complete or an incomplete rupture.

TABLE IV.—FINDINGS AT OPERATION

Incomplete rupture of uterus:	Hernia-like bulging of the uterine wall at the point of rupture.
Complete rupture (all layers)	Blood and clots
	Fetus free in abdomen
	Fetus and placenta free in abdomen

As the prognosis is always serious, since the maternal mortality is around 50% and the fetal mortality 100%, we suggest the following procedure.

TABLE V.—PROCEDURE TO FOLLOW

1. *Predisposed women*
   - Previous Cæsarean section
   - Previous ligamento or hysteropexy
   - Once a Cæsarean always a Cæsarean (Except for a very small number of cases)
2. *Actual rupture*
   - Only well equipped centres can save mother and child
   - (*a*) High speed transfusions and shock therapy
   - (*b*) prompt operation
     - Uterine suture (limited number) or
     - Hysterectomy (majority)

SUMMARY AND CONCLUSIONS

1. Presentation of a case of spontaneous uterine rupture with survival.

2. Spontaneous ruptures make up 1/10 of all uterine ruptures.

3. Symptoms can be dramatic (complete rupture) or minimal (incomplete).

4. High speed transfusion and immediate operation save mothers.

5. Hysterectomy must be performed instead of uterine suture.

6. Multiparity in itself should not be considered as a predisposition to rupture. As proof of this, its low incidence in French-Canadian women, reputed for their large families.

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ANTITHYROID DRUGS IN THE TREATMENT OF HYPERTHYROIDISM

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FOR many years the treatment of hyperthyroidism centred around the surgical removal of the greater part of the thyroid gland after preliminary preparation with iodine, rest, dietary and other symptomatic measures. Such treatment attained a high degree of safety and efficiency but was not ideal in that, in addition to occasional purely surgical complications, a significant proportion of cases developed relapses. In the past five years two new methods of approach have been introduced, namely antithyroid drugs and radioactive iodine, which will probably have a considerable place in the treatment of hyperthyroidism. This communication deals wth the antithyroid drugs, thiouracil and propyl-thiouracil and their use in this disease.

Four years' experience has proved beyond doubt that thiouracil effectively inhibits the elaboration of the thyroid hormone, probably by a specific block of the uptake of iodine in the hormone molecule. By reason of this property it relieves all the effects observed in human hyperthyroidism which can be directly attributed to the excess of thyroid hormone. When thiouracil is administered in adequate dosage over a sufficiently long period of time, practically every case of thyrotoxicosis whether Graves' disease or toxic nodular goitre, returns at least to a state of normal metabolism and if maximal doses are continued myxœdema will eventually result. If, however, smaller or maintenance doses are given, it has proved possible to maintain the metabolic state at normal levels. The optimum dose for complete inhibition with thiouracil is approximately 600 mgm. (9 gr.) given in three equally divided doses throughout the day and the average maintenance dose is between 100 and 300 mgm. It may be said that the effect of thiouracil is to produce a chemical thyroidectomy, the degree of which may be controlled by the dose but that this effect only persists while the drug is administered. The effect of thiouracil is not immediate, and depending on certain factors, it requires from two to three months to accomplish the desired result. Table I enumerates the main factors which have been shown to enhance or delay the rate of response.

TABLE I.

Factors hastening response:	*Factors delaying response:*
1. Short duration of symptoms.	1. Long standing symptoms.
2. Mild hyperthyroidism.	2. Severe hyperthyroidism.
3. ? Diffuse glandular enlargement.	3. Nodular or adenomatous enlargement.
4. Minimal enlargement.	4. Marked enlargement.
5. Concomitant iodine (?).	5. Preliminary iodine.
6. Adjuvant treatment (Rest, diet, etc.)	6. No adjuvant treatment.

As a direct result of the depressed thyroid function produced by thiouracil structural changes occur in the gland itself which are mediated through the pituitary. These consist of a marked hyperplasia with loss of colloid substance and increased vascularity, essentially the changes of Graves' disease itself. Such changes have been found to complicate the surgeon's task but can be reversed by iodine administration, after withdrawal of thiouracil. Thiouracil does not relieve the ocular changes of hyperthyroidism and there are some who believe that owing to increased pituitary thyrotropic activity exophthalmos may even be aggravated. Clinical evidence on this point is not, however, convincing and it is probable that

it has no more deleterious effect on exophthalmos than thyroidectomy.

Such are the actions of thiouracil which would seem to have distinct advantages over a major operative procedure, but on the other side of the ledger are definite disadvantages. The long term results of thiouracil treatment will be discussed later, but more important is the fact that it has proved to exhibit important toxic manifestations.

TABLE II.
TYPE AND INCIDENCE OF TOXIC REACTIONS TO THIOURACIL

Agranulocytosis	2 to 2.5%
Mortality 20 to 30%	0.5%
Leucopenia (less than 3,000)	3 to 5.0%
Drug fever	3 to 5.0%
Drug eruption	3.0%
Diffuse glandular enlargement	RARE BUT
Salivary gland enlargement	IMPORTANT
Oral infection (gingivitis and stomatitis)	
Thrombocytopenia	
Gastro-intestinal disturbances	Relatively
Vertigo	unimportant
Total incidence of reactions necessitating cessation of treatment	Approximately 10%

In Table II are listed the more important toxic reactions to thiouracil and their approximate incidence as obtained from several series and surveys in the literature. The striking feature of the table is that one patient in every forty to fifty treated developed severe neutropenia in spite of careful clinical observation and frequently repeated blood counts and of these, one-fifth to one-quarter died of agranulocytosis. From this complication alone, the overall mortality from thiouracil equals or surpasses the mortality of thyroidectomy in competent hands. Furthermore, of those treated with thiouracil one patient in ten developed a reaction sufficiently severe to preclude further administration. Although the peak incidence of reactions occurs in the first eight to ten weeks when the larger doses are required, careful and frequent clinical observation must be continued for as long as the drug is being given, as reactions have been observed at any time. Frequent leucocyte counts and smears may detect early leucopenia but have not proved an adequate means of predicting agranulocytosis which may, and commonly does, appear with dramatic suddenness so that in addition patients must be warned to be on the outlook for any febrile illness, sore throat or skin eruption and to stop the drug forthwith and report. It is the responsibility of the physician who embarks a patient on a course of thiouracil to insist on frequent clinical examinations and to prevent the patient continuing self medication without supervision.

Thiouracil has proved to carry about equal risks to thyroidectomy if all cases of hyperthyroidism are considered as a whole, but it has definitely greater risks in the average mild or moderate case in which operative mortality is rare and the ultimate result eminently satisfactory. On the other hand, when used as a preliminary to operation it has materially reduced the risk in cases of severe thyrotoxicosis and in those with cardiac failure. In fact, adequate preliminary treatment with thiouracil until the basal metabolic rate has become normal, followed by preoperative iodine has rendered the prognosis of operation in this group the same as for mild cases or non-toxic goitre. Because of its toxicity most have agreed that thiouracil was best reserved for specific purposes, namely:

(1) Recurrent thyrotoxicosis following one or more adequate thyroidectomies, in which instance prolonged administration was usually necessary; in other words, in the relatively small group in which thyroidectomy apparently failed. (2) In patients with severe toxicity, cardiac failure or other complication such as iodine resistance which made immediate operation even after iodine preparation hazardous, thiouracil should be used as a preliminary to operation until the basal metabolic rate is normal. Such use of thiouracil has probably completely done away with the necessity for multiple operations. (3) Finally, it is justified to use it as the definitive method of treatment in patients in whom operation was refused or otherwise contraindicated.

Ever since the toxicity of thiouracil was appreciated the search for equally effective and non-toxic alternative drugs has continued. A number of substitutes and allied compounds and several unrelated substances have received trial, but recently what appears to be the most promising, 6-Propylthiouracil has been made generally available. Propylthiouracil would appear to be as effective as thiouracil in about one-quarter to one-third the dose, although possibly the effect is not so rapid. Rapidly accumulating clinical experience indicates that when given in doses of 150 mgm. daily it controls about 60% but larger doses of 200 to 300

mgm. may be required. Smaller doses have been effective in some cases but the effect of such doses is unpredictable. The effective maintenance dose may be as little as 75 mgm. a day or less.

What of its toxicity? It is certainly less toxic than thiouracil. Occasional drug fever and skin eruptions have been observed and although no fatal case of agranulocytosis has been reported, one non-fatal case has been observed, an indication that it is at least potentially dangerous. The total incidence of important reactions in patients receiving 150 mgm. daily is probably not more than 1 to 2% and neutropenia proportionally less. If further experience bears this out, the dangers of propylthiouracil will be sufficiently remote to justify its wider application.

In considering broadening the uses of propylthiouracil or any other antithyroid drug, the experience that has been gained from past knowledge of the nature and natural history of hyperthyroidism and from the use of thiouracil serve as the only guides. The stimulus or stimuli which give rise to thyroid hyperfunction are still unknown but there has long been the belief which seems to be gaining favour that the thyroid itself is not primarily responsible but merely the end organ. Therefore, the same criticism that has been applied to thyroidectomy can be applied to antithyroid drugs and also to radioactive iodine, namely that direct attack on the thyroid does not remove the fundamental cause. Nevertheless, while such cause or causes are not known such indirect attack is justified by its result. Furthermore it is known that hyperthyroidism is usually a cyclic disorder with periods of exacerbation and remission and if the subject survives the ill effects of the disease it may become inactive in the course of time. The problem at present, then, would seem to resolve itself into which method should be applied in each case, bearing in mind the risks involved, and the individual social and economic problems in each case. For example, the problem of treatment differs in an urban housewife and an isolated farmer even though the type of disease, the computed risks and expected results may be identical. The consideration of such factors cannot be entered into in this discussion, and it will be confined to the clinical aspects of the disease itself, in the light of experience to date.

Experience with the prolonged use of thiouracil has shown that antithyroid drugs will induce remissions for at least as long as the drug is taken, and that with a proper maintenance dose which must be determined for each case, the patient remains in a state of normal health. Such a dose may vary according to the activity of the underlying but suppressed disease and in fact, the dose required serves as a rough index of this activity. While properly maintained the patients may lead a normal life in every way they should be kept under occasional medical supervision, not only to avert toxic reactions but to gauge the progress of the disease. While it is not yet proved, it would appear that heart failure and cardiac arrhythmias respond as well to thiouracil as to operation, and there is no contraindication to the use of quinidine or other cardiac drugs. Neither pregnancy nor the possibility of pregnancy contraindicate the use of propylthiouracil, provided myxœdema is averted, as it is the myxœdema and not the drug which may endanger the fetus. There is no clinical evidence as yet that the hyperplasia produced in the thyroid bears any relationship to the development of carcinoma of the thyroid and such a relationship, though not finally excluded must be a very remote possibility. However, drug therapy must be continued for long periods of time.

My own experience with prolonged administration has not been large but from the results reported in the literature the relapse rate, even after nine months' treatment or longer, is approximately 50%, within five to six months of cessation of treatment. Relapses after this period of freedom are uncommon but do occur. With repeated courses this incidence may be further reduced but it will be seen that this may involve up to several years' continuous drug therapy. It would appear, however, that selection of cases may improve this picture. Cases with pressure symptoms should be treated with thyroidectomy as soon as possible. If hyperthyroidism is severe, preoperative propylthiouracil is indicated. Cases with marked enlargement of the thyroid show a high incidence of relapse even after prolonged treatment and will probably require thyroidectomy which is best not deferred. For some reason the incidence of relapse in males is very high and propylthiouracil is unlikely to produce prolonged recovery.

In the present state of our knowledge of antithyroid drugs and in particular, propylthiouracil, I believe they may best be employed in recurrences following thyroidectomy, as a preoperative treatment for severe cases which will require operation later, and as a definitive method of treatment in cases without marked thyroid enlargement, especially women, provided such patients realize the necessity and are sufficiently co-operative to remain under surveillance at weekly intervals for the first two to three months and one to two month intervals thereafter and are conversant with the toxic potentialities of the drug. Prolonged self-medication without such surveillance is not justified. Patients with marked enlargement, especially men and possibly those with large or multiple adenomata will probably ultimately require thyroidectomy which should not be deferred once the disease is controlled. Although the low incidence of toxic reactions to propylthiouracil is encouraging, it should not relax vigilance. Only time can tell what the ultimate place of these drugs will be in the management of hyperthyroidism. I personally do not feel that they constitute the final answer, and that they will entirely replace operation. Possibly radioactive iodine may change the picture in the future but in the meantime they provide a distinct forward step in the safe and effective control of thyrotoxicosis, as a preoperative measure, and will probably render operation unnecessary in a fair proportion of selected cases.

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The Royal College of Physicians and Surgeons of Canada

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THE CANADIAN MEDICAL ASSOCIATION

Editorial Offices—3640 University Street, Montreal

(Information regarding contributions and advertising will be found on the second page following the reading material.)

EDITORIAL

PHYSICIANS' SERVICES, INCORPORATED

THE profession generally will watch with great interest and solicitude, the progress of Physicians' Services, Incorporated, the organization set up in Ontario by the Ontario Medical Association for prepaid medical care in that Province. Dr. C. C. White, the President of the Ontario Medical Association, writes as follows in the *Ontario Medical Review* (October, 1947).

"The granting of the charter to the Physicians' Services, Incorporated is both a satisfaction and a challenge to the medical profession of Ontario. After many years of study and debate the Association has the satisfaction of seeing its considered decision come to fruition. The challenge is in the functioning of the new organization and its success in providing a solution to the medico-economic problems confronting the people and therefore ourselves. Upon the degree of co-operation, fairness and sense of personal responsibility of each member of the profession depends the success of the scheme. Seldom has such a loosely organized, individualistically minded group ever been challenged to so change habits of thought and conduct and to learn a new way of life, as it were,—for the new scheme implies just that—in the conduct of 'the business of private practice'. The professional side will not be greatly affected. The removal of an economic barrier between patient and doctor should result, if in any change, in a more satisfactory life and an overall improvement in professional works. However, on the business side the change will be real and the effort required to re-adjust methods and procedure and habit of thought will be considerable. But the end will be worth all the effort for in this we have an opportunity to provide the great mass of the people with our services, which they want, at a price they can afford, and by a method of payment which is convenient and suitable. To the profession it offers an opportunity to retain control of our business and professional affairs, and to leave undisturbed the patient-doctor relationship.

"In the broader field of human relations, it presents an opportunity to the profession to make a real contribution to the cause of personal freedom and responsibility, on which depends everything we hold dear in civilization. Let us realize the opportunity for what it is, and may we not 'muff it'."

We sincerely hope the response will be worthy of the earnestness of his appeal.

PROGRAM ANNOUNCEMENT FOR THE ANNUAL MEETING

THE Seventy-ninth Annual Meeting of the Canadian Medical Association will be held in Toronto during the week of June 21, 1948, in conjunction with the Sixty-eighth Annual Meeting of the Ontario Division.

Any member of the Association who desires to contribute to the program is invited to notify the Central Program Committee by writing to the General Secretary of the Association at 135 St. Clair Avenue West, Toronto 5, not later than December 20, 1947.

Plans for the meeting are well under way and a large attendance is expected.

EDITORIAL COMMENTS

The Canadian Rheumatism and Arthritis Society

There has been for some time an increasing public demand for a co-ordinated effort to deal with the problem of rheumatic disease. Our Association has a section devoted to this subject, but there is need also for an organization with a wider scope. This new body is now in process of formation and is to bear the name of "The Canadian Rheumatism and Arthritis

Society''. It had its inception at a meeting recently arranged through the Minister of Health and Welfare, at which representatives from several interested groups were present, including the universities and national research, public health, and voluntary organizations such as our own, and the dental and nursing professions, with several others. The Department of Veterans' Affairs was also represented.

The objects of the Society are designedly as wide as possible, namely: prevention; diagnosis; treatment; training of personnel; research; public education; raising of funds.

An interim committee has been formed under the presidency of Dr. Wallace Graham of Toronto and the drawing up of the necessary charter and by-laws is now being carried out.

It is obvious that the Society must have a substantial lay membership; in arranging for this it will probably follow the main pattern of certain other national voluntary organizations, such as the Canadian Red Cross, the Canadian Cancer Society, the Canadian Tuberculosis Association and many others. All the details of organization have yet to be settled, such as the location of head office, the appointment of an organizing secretary, the selection of the directors, and so on. A problem of such magnitude as rheumatic disease calls for the widest possible effort, and our Association is ready to do its full share in promoting the aims and objects of this Society.

The Victorian Order of Nurses

The Victorian Order of Nurses for Canada has celebrated its 50th anniversary by publishing a history of the Order.* But this fifty years is only a conveniently marked period. The V.O.N. embodies all the best age-long traditions of nursing; sympathy, energy, and solicitude for those in distress. To the sick person it matters little whether these ideals are followed in a hospital or on visits to the home; but it matters very much that the V.O.N. *has* brought them to the home.

The Sisters of Charity of Montreal (the Grey Nuns) followed by the Sisters of Providence did true district nursing, visiting in pairs: the founder of the Grey Nuns, Madame d'Youville of Montreal, conceived the idea of the Order from St. Francis. But the V.O.N. has an *esprit de corps* all its own. In 1897 their founder and first President, Lady Aberdeen began her campaign for a memorial fund to celebrate the Queen's Diamond Jubilee by forming in Canada an organization of visiting nurses similar to the Queen's Nurses who had been organized at the time of the celebration of Queen Victoria's first Jubilee in 1887. By her efforts the V.O.N. eventually received its charter. Florence Nightingale's influence is directly evident in the V.O.N. since she formulated the rules and training for the Queen's Nurses whose methods have been adapted to the Canadian scene. The two organizations are independent of each other, but pursue the same ideals.

But let no one think that the V.O.N. merely sprang up. Perhaps the most interesting part of the extremely attractive history by Mr. Gibbon is the account of the efforts to gain the support of the medical profession for the Order. The best that can be said is that the opposition did not last long, and that the conversion has been complete.

The V.O.N. has shared in every aspect of Canadian history in the last 50 years; from the Klondyke gold rush in 1898, ''The Trail of '98'', the effects on Canada of two wars, a depression, and epidemics. It has grown steadily and is now a great national Order, playing a vital part in the public health of Canada. Mr. Gibbon has given an interesting and valuable account of its growth.

* Victorian Order of Nurses for Canada, John Murray Gibbon, 121 pp., illust. $2.00. The Southam Publishing Company, Montreal, 1947.

Scientific Exhibits

Recognizing the outstanding educational value of scientific exhibits, it has been decided to resume this feature at the forthcoming annual meeting of the Canadian Medical Association which will be held in Toronto during the week of June 21, 1948.

This preliminary announcement is intended to advise members who have material suitable for visual display that a scientific exhibit will be held. Prospective exhibitors should communicate with the General Secretary, C.M.A., 135 St. Clair Ave. West, Toronto, for further details.

SECOND ATTACKS OF POLIOMYELITIS.—It is commonly assumed that an attack of poliomyelitis with paralysis confers a life-long immunity. In 1930, Still noted that what appeared to be second attacks of the acute disease were occasionally reported, but he regarded many of them, occurring within four months of the initial illness, as recrudescences of the original infection to be distinguished from true second attacks due to reinfection, which were distinctly rare. Still found that between four months and two years after the first attack there was an interval during which second attacks had never been recorded, and he therefore postulated about two years as the probable maximum duration of the immunity resulting from an acute attack. Of 2 cases recorded by Colonel Lipscomb, in one the second illness occurred about four months after the initial attack and may possibly have been a recrudescence; in the other the second attack was separated from the first by nine years and reinfection is probable. In 1938 Fischer and Stillerman collected 13 authentic instances of second attacks arising two to twenty-four years after the first. They added a further 4 examples observed during the 1935 outbreak in New York. After making a statistical study of this outbreak and considering the available data they concluded that the admittedly very low rate of incidence of second attacks was not evidence that any immunity was necessarily conferred by an attack of poliomyelitis.—*The Lancet*, p. 551, October 11, 1947.

SPECIAL ARTICLE

THE REFUGEE DOCTOR

As some interest has been displayed by the Canadian public in the Refugee Doctors now residing in Displaced Persons camps in Europe, the Canadian Medical Association desires to make a statement on the subject.

The World Health Organization which has been established by the United Nations, and the World Medical Association, are deeply conscious of the problems confronting displaced persons, particularly that group representing the healing arts, and have begun studies looking to the ultimate p'acement of these persons. In order to know the magnitude of the problem and all details associated with it, it was necessary that a survey be made. At the last meeting of the Interim Commission of the World Health Organization held in Geneva during the first two weeks of September, 1947, the following statistics were presented:

PROFESSIONAL PERSONS LOCATED IN DISPLACED PERSONS CAMPS

Category	Germany		Austria		Italy	Middle East	India	Shanghai	Total
	U.S. Zone	British Zone	U.S. Zone	British Zone					
Dentists	750	150	15	3	10	..	..	30	958
Physicians and Surgeons	881	413	139	17	45	6	..	111	1,612
Ophthalmologists	24	9	2	2	2	..	..	1	40
Pædiatricians	72	33	1	..	70	..	..	..	176
Nurses, including registered, practical and training	2,077	1,452	not known	not known	246	..	..	..	3,775

It was recognized that it would be necessary to have a careful inventory of each person, covering the following points: What professional training have they had? What proficiency do they now possess? When were they last engaged in their professional pursuits? What is the state of their physical, mental and moral health? What are the reasons which prevent them from returning to their native land? If given a preference, where would they like to live?

It should be emphasized that every individual concerned must be dealt with separately. It is already known that many of these professional people have not pursued their calling for long periods of time, some having been out of practice for many years. Before they are ready to resume professional duties it will be necessary for many of them to take postgraduate or refresher courses. This presents a problem because available teaching facilities all over the world are taxed to the limit, bearing in mind the destruction of many of the institutions of learning during the war and the very considerable backlog of students whose training was interrupted or delayed for several years due to war service. But, while the difficulties are great and sometimes appear overwhelming, the World Health Organization and the World Medical Association propose to leave no stones unturned to do all in their power to assist these professional persons to become rehabilitated and self-sustaining citizens, once again established in homes. The World Medical Association which came into being in September, 1947, and which is composed of 48 national medical associations representing upwards of 500,000 doctors, has begun a world-wide survey which includes in its scope an inventory of medical supply and demand. In this connection, the following information has been gleaned from various sources believed to be authentic:

Ethiopia, a country of twelve million persons, reports that, at the close of the war, they had not a native doctor or nurse, as all had been liquidated by the enemy.

Czechoslovakia reports that of the 12,000 doctors dwelling in that country when the war began, 60% (over 7,000 in number) are dead or missing.

China, with over four hundred million people, has less than 12,000 trained doctors. During the war, the Chinese army was served by 30,000 medical officers, only 7% of whom had had medical training.

India reports a total medical registration of 40,000 to serve more than four hundred million people and the need for doctors in that country is very great.

Liberia, a country of 2,600,000 people, has one doctor.

When the survey is completed, it is fully expected that there will be available a clear picture of medical conditions and needs of every part of the world, save perhaps in Russia, which is not at the moment a member of the World Medical Association.

The Association will have in its possession records, advice, and suggestions with respect to the medical needs of the people such as have never before been assembled. This survey is being made just as rapidly as human endeavour can be marshalled to do it and it would appear that these two great organizations working along

parallel lines—the World Health Organzation representing many nations of the world, and the World Medical Association representing the doctors of the world, will be in a position to render intelligent advice and guidance to a group of people who at the moment are homeless and most anxious to begin life over again.

CONDITIONS IN CANADA

The Canadian Medical Association, along with all the other national medical associations, has been asked to reply to a very complete and comprehensive questionnaire. Fortunately, an alert and active National Department of Health in Canada has kept up to date the survey of the healing arts which was made by the Canadian Medical Procurement and Assignment Board during the war. One does not have to engage in guess work about Canada's medical needs. We know where our 13,000 doctors are located. We know what areas are said to be medically over-populated as well as those areas which are under-populated. We know the places in which there are no doctors and also the areas where it is believed a doctor could not make a living without subsidization. We know how many medical students are in training and the number of recent graduates who are taking advanced work or rehabilitating themselves after war service. Stated in other words, we have an over-all picture of medical supply and demand in Canada, upon which intelligent advice can be given to those areas seeking doctors and to doctors seeking locations.

It is the considered opinion of medical authorities in Canada that our medical schools, of which we now have nine, graduating an average of 600 doctors a year, can take care of Canada's medical needs allowing for losses due to death and emigration, and also making allowance for the newcomers who from time to time come to our shores. Furthermore, the University of Saskatchewan is well on the way towards providing a complete medical curriculum which will add upwards of 50 medical graduates a year to our total output. Ottawa University is developing a faculty of medicine, and the University of British Columbia is seriously considering the establishment of a medical school which, when functioning, will further increase our yearly output of doctors. On the whole, it may be said, looking at our country in relation not only to its medical needs but to the needs of other countries of the world, that Canada is reasonably well off for doctors.

Now it must be admitted that the distribution of Canada's doctors might be improved. The larger urban areas may have more doctors than they need, although that is a statement which must not be made too dogmatically; and the rural areas, at least some of them, have not enough doctors. The authorities concerned in this matter are mindful of this situation and are considering ways and means to improve it. In the opinion of the Canadian Medical Association, rural areas which can support a doctor and which make life for the doctor satisfactory by providing him with hospital facilities and suitable working conditions will ultimately find a solution of their problem. The answer to the lack of medical care in the rural communities does not lie in forcing, co-opting or importing a doctor and setting him down there. He must have satisfactory working conditions and be able to make a decent living for himself and his family. But the refugee doctor may not wish to go to the country. It may come as a surprise to some readers to know that since the beginning of the war, 60 refugee doctors have been admitted to Canada. Of this number, only five are now engaged in rural practice. It has been suggested by some members of the public that the refugee doctor be imported on the understanding that he must reside in the country. Has Canada ever before compelled any of her new citizens to live in a circumscribed part of the country? What a hue and cry there would be if medical or other authorities devised regulations compelling any doctor to live in the country if that doctor decided that he wished to live elsewhere. No doubt many of the refugee doctors might like to live in the country, but, on the facts as we now have them, there can be no guarantee that the problem of providing medical care to rural Canada would be solved by importing doctors from Europe.

Recognizing the dire need for doctors in many countries of the world, as has already been shown in this memorandum, on what grounds could it be urged that doctors in Displaced Persons Camps in Europe be brought to Canada? A statement has been made that we would have no more right to bring doctors from Europe to Canada than we would have to ask Europe to send us food parcels.

It has been suggested that if Canada does not need these doctors we might at least bring them here for postgraduate studies, looking to their professional rehabilitation. Any such suggestion must allow for the cost of such an ambitious program. Cognizance must be taken of the fact that all available facilities for training doctors in Canada are now taxed to the limit and the Universities and places of medical teaching state that it would be found extremely difficult if not impossible to place any appreciable number of refugee doctors in hospitals for training.

The Canadian Medical Association has pointed out more than once that medical standards in Canada are high, perhaps as high as are to be found in any country of the world, and we can not afford to dilute that standard with medical imports of lower standards or imports who have been out of practice for such a lengthy period that they are not now competent to practise their art without being professionally rehabili-

tated. It is not suggested that all refugee doctors are rusty nor that any high percentage of them are poorly trained, but the public interest will not permit of a double standard of qualification and all physicians, whether trained in Canada or elsewhere, must measure up to the licensing requirements which are in effect. A doctor whose medical degree has been obtained anywhere in the world can establish himself in a Canadian province provided that he enters the country legally, obtains an enabling certificate, passes the examinations of the Medical Council of Canada and registers this qualification with a provincial licensing body.

But let it not be said that the Canadian Medical Association is uncharitable or unkindly in its thinking towards these professional brethren who find themselves in such a sorry plight. The Association is glad of the opportunity of rendering any assistance in its power to the World Health Organization and the World Medical Association in the study of this problem, looking to a happy and satisfactory solution.

MEN and BOOKS

BARBED WIRE HUMOUR*

J. N. Crawford, M.D.

Winnipeg, Man.

In the fall of 1941, two regiments of Canadian soldiers were sent to reinforce the garrison at Hong Kong. Four medical offiicers, of whom I was one, accompanied this force. On December 8 the garrison was attacked by the Japanese. For seventeen days we struggled against overwhelming odds and on Christmas Day we were forced to surrender. From then until August, 1945, we were under the domination of the Japanese Army as prisoners of war.

The stories which I propose to tell you today are reminiscences of that period of internment. Many of you have heard these stories at least once before. The fact that you seem willing to listen to them again encourages me to believe that they are perhaps more interesting than I think them to be. I do not propose to dwell upon the unpleasant aspects of this period. Unpleasantness there was, and plenty of it, but with the passage of time the memory of this has become dim and now the most vivid things are those which made us laugh and which in fact preserved our sanity.

At the beginning of internment we were bundled into a camp with very sketchy facilities for hygiene and no facilities whatever for the medical care of the sick. It seemed to us that our captors, having shoved medical officers into the camp along with the other prisoners, took no further responsibility with respect to the care of the sick and wounded. This attitude was modified only slightly during the subsequent years of captivity. As a result, any assistance which we were able to give the sick in camp was in proportion to the ingenuity which was shown in the provision of a place to work in and tools with which to work. For such ingenuity, full credit must be given to the many mechanics and artisans who were our fellow prisoners. I remember in particular, three examples of this spirit of "make do".

Some months of very bad diet in camp produced a deficiency syndrome, one feature of which was a loss of sensation. We wanted a tuning fork with which to test vibratory sensation, and had none. We described what we wanted to the mechanics of the ordnance corps, and they manufactured a tuning fork. True, it was so big that it took nearly two men and a boy to hold it, and it almost shook the leg off the patient to whom it was applied, but it worked. We were reasonably certain that a patient who could not feel the vibrations of this monstrosity had more than slight damage to his nervous pathways.

Somewhat later, progression of this same deficiency syndrome produced damage to the ocular nerve. We wanted to map out peripheral visual fields in these patients, and decided to manufacture a perimeter. It was a long time since any of us had seen, much less used, a perimeter, but after much brain beating we managed to produce a sketch of what we wanted. The mechanics thereupon produced, out of bits of wood, tin and wire, a perimeter which, *mirabile dictu*, worked. Unfortunately, we had neglected one factor, that is that we were dealing with a central scotoma, and that the patient could not fix on the central target of the perimeter. But that was not the fault of the mechanics, and if anyone wants a good, workable perimeter, cheap, I know where there is one in Hong Kong.

I was particularly proud of the operating room equipment which I had at the time we were relieved. Our early surgery was done on a wooden bench, using a pocket case of instruments which were sterilized on the kitchen stove. Surgery had to be done in daylight, as we had no other means of illumination. Little by little, we were able to improve on this situation, and at the time we were relieved, I was the proud possessor of a reasonably equipped operating theatre. We had a table that was adjustable to three positions, with a built-in tiltometer for use with spinal anæsthetics. We also had a "shadowless" (or almost so) operating light, in which the source of illumination was a battery of twenty-four wicks, burning peanut oil. We also had a sterilizer in the theatre, which operated from an outside boiler.

* A luncheon address, at the Annual Meeting in Winnipeg, June 26, 1947.

All this was manufactured in camp by our own mechanics, who used such material as they could find, "scrounge" or just plain steal.

The question of the supply of medical equipment, instruments and drugs was one with which we were always faced. Very little, much less than we required, was supplied to us through official Japanese channels, and we were early forced to obtain things "illegally" through the medium of our Japanese guards. There were various ways of persuading a Jap to do business with us. My first success along this line was with a Japanese sergeant who had read some Shakespeare at school, and who was anxious to improve his English. In return for supplies of medicines, I spent long hours reading "Romeo and Juliet" to him, or listening to him read and correcting his accent. We never quite got around to doing it, but on looking back I feel that we should have had some sort of fixed tariff; something like a hundred tablets of sulfapyridine for every scene I read to him, and a thousand for every scene I listened to. Maybe ten thousand, if I had to stay awake while he read.

Fortunately, the Japanese medical officer in the area was about as rough to his own troops as he was to us, and the guards hated to go to him for treatment. They much preferred to consult us. Venereal disease control in the Japanese Army was not very efficient, and there were few days when we were not requested by a guard to do something about his primary chancre or roaring discharge. For such treatment, the Jap had to supply his own medicine, purchased on our advice. Depending upon whether our own troops were particularly plagued by malaria or beriberi at the time, it was surprising how much quinine or thiamine was necessary to treat syphilis in a Japanese soldier, in addition of course, to a small amount of intravenous arsenical. We were always pleased to treat a chancre in a Jap guard. Invariably, when the lesion had cleared up after a few "shots", he would refuse further treatment, and insist that he was now cured. We were happy to see him come, as he paid us well in the form of drugs that we needed for our own troops, and we were happy to see him go so quickly, as we fervently hoped that ten years or so later he would be a lovely case of G.P.I. Perhaps we were not strictly following the ethical teaching of our profession, but the Japs were pleased, and the College of Physicians and Surgeons was not watching us.

One way and another we had a lot of fun with the Japs, particularly in the matter of the routine searches of the camp for contraband. The Jap has a one-track mind. If he is searching for radios, for example, he will ignore tunnelling tools, even though they are in plain sight. If he is searching for tunnelling tools, he will ignore a radio. On the occasion of every search, except one, we were given adequate warning by our "tame guards" not only as to the time of the forthcoming search but as to the specific articles which would be looked for. Such articles we hid carefully.

Documents of all sorts were particularly suspected by the Japs. We had a mass of medical records which we were most anxious to retain. After several sad experiences, when some records were found and confiscated, we resolved to adopt the principle that what is most obvious is best hidden. The Japanese controlled an orderly room which was quite swamped with their own particular form of "bumph", which was naturally beyond suspicion. We therefore placed our medical documents in this orderly room, in a casual sort of way, and due to the location, these took on the aura of innocence. They were never disturbed, and when we were relieved, we were able to recover most of the information we had gathered. We should thank the Japanese for the protection they gave material which was subsequently used as evidence against them.

At one time, while we still had electricity in the camp, I was able to persuade the camp commandant that I needed a small electric plate for the purpose of sterilizing instruments. However, the "Kempitai" or "Gestapo" who conducted the searches had given no authority for the retention of such a dangerous bit of apparatus. As a result, on the occasion of every search, my electric plate was removed, and dumped in the camp commandant's office along with the other contraband. Afterward, I would interview the camp commandant and after much argument would be able to persuade him that I should have the plate back. The camp guards soon became conditioned to this drill. They would see the heater go out in the hands of the gestapo, and then would see me carry it back. Eventually, I grew tired of these repeated arguments with the commandant, and decided to shorten up the process, if I could. Thereafter, as the gestapo would march the heater up to the commandant's office, I would follow them. When they had dumped the heater on the pile of contraband, and had gone, I would pick it up, and march it home again. This saved a lot of time, and was a lovely game while it lasted. It only ended when the electricity was cut off for good, and the heater had no more use.

Most of our guards were Formosan boys, and I think that some of them took a genuine liking to us. At least they did not dislike us as much as they did their Japanese officers. As the war in the Far East drew near its inevitable close, many of them read the writing on the wall and deserted. One night in August, 1945, one of these Formosans with whom I had had dealings, came into my room and woke me up. He asked me where I was going after the war, and I said that of course I was going to Canada. He then said that he was an "ichi ban" (the Japanese term for number one, or excellent) houseboy, and asked me if I would take him with me in

that capacity. When I said that I could not do that, he said "Okay goodbye" and disappeared into the night, forever. When I arrived home and discovered the scarcity of domestic servants in Canada, I regretted that I had not brought him with me. I could have classed him, for customs purposes, as "souvenirs". He would have been useful around the house, and every morning when he brought me morning tea, I could have kicked him in his lovely artificial gold teeth, just for old time's sake.

I was frequently impressed by the methods of Japanese medicine. They are firm believers in the efficacy of barbed wire. In order to control an epidemic of dysentery for example, it is only necessary to wire off an isolation area, employing a single strand of barbed wire. For a more virulent infection, such as diphtheria, stronger measures are necessary, and two strands of wire must be used.

Speaking of diphtheria, I am reminded of a Japanese remedy that did work, at least temporarily. We were suffering a severe epidemic of diphtheria at the time, and no anti-diphtheritic serum was available. We were showing a horrible death rate. Finally the Japanese did provide a small amount of serum, though not nearly enough for the number of cases we had. The following day we had three more deaths. The Japs took a very poor view of this. They had provided the serum we had asked for, and men still died. Therefore, the responsibility of further deaths was clearly ours. Thereupon they lined up my medical orderlies and myself, and gave us a bit of a belting. This caused me a certain amount of chagrin, but what was equally upsetting was the fact that during the succeeding twenty-four hours, there were no further deaths. Clear proof of the efficacy of Japanese methods of treatment!

The position of a medical officer in a prisoner of war camp under such circumstances is peculiar. He is the only officer who can be of any noticeable assistance to the men, and the men turn to him with all sorts of problems which are quite outside the medical field. If he shows an ordinary interest in his troops he is revered, and if he shows more than an ordinary interest he stands in great peril of being deified. This is an uncomfortable rôle, that becomes most of us badly. On the other hand, the height of the pedestal upon which he stands makes him the obvious butt of the crude humour of the camp. One of the medical officers of our camp was a diligent, scholarly type, inclined to the classical professorial sort of absentmindedness. The stories told of him are legion, but two stick in my memory.

He was one day examining a man in the medical inspection room. The patient suffered the very common deficiency syndrome, and complained among other things, of being numb from the waist down. The doctor listened to his story, and then began to map out the anæsthetic area, using a pin. He was called away in the middle of the examination on an urgent call, and forgot to return. The next day, the same patient appeared. The doctor had quite forgotten that he had ever seen him before, took his history, and then began fumbling for his pin. Said the patient, turning his stern to the doctor, "Is this what you're looking for, doctor? You left it stuck in me yesterday."

The other story about the same man concerns him making rounds in his hospital ward one day. He was listening to the complaints of a poor emaciated devil lying in bed. Said the patient, "I've been very constipated, doctor. My stool is just like wood." "Really", said our doctor. "Really. How very interesting. What kind of wood?"

These stories are probably not true, but they might have been true. I don't really care very much. I only know that I shall never see a safety pin or a cathartic pill again, but that I will remember my absentminded friend with a great deal of pleasure.

I think that today I am bold enough to tell a story on myself, which has never been told except to a very small select group who were "on the inside". The reason for this unusual reticence is that the story tells of what is, as far as I know, my only venture from the stony path of rectitude into the flowery fields of major crime. We were allowed by the Japanese, in order to eke out our meagre rations, to operate what was euphemistically called a "camp farm". The catch in this was that the camp and the farm were situated on reclaimed land, sand and gravel on which, without fertilizer, nothing would grow. Fertilizer was essential, but where to get it? There were no horses in Hong Kong, the natives had eaten them long since. On the other hand, there was no plumbing in camp, and there were a thousand inmates with a whole battery of bucket latrines. The connection between our need of fertilizer and the providential primitive hygienic facilities was obvious. I, and the other medical officers, long waged a losing battle on the question of fertilizer for the farm. It was difficult to argue against the age old customs of several hundred million Chinese, and anyway, the whole camp was thoroughly infected with ascaris, bacillary and amœbic dysentery. We didn't really have a leg to stand on, and the farmers beat us, hands down. I, like everyone else, ate my share of the produce from this beautifully fertilized farm, and liked it. And now comes the confession. My roommates, who were two medical officers, and I, had a little private garden, outside our door. We planted tomatoes, and carrots and such, and I had a little gardenia bush of which I was inordinately proud. But the soil was harsh, and most unfertile. Our tomatoes leafed, but flowered not; our carrots topped, but bottomed not, or whatever it is that carrots do; and my little gardenia bush hung its pretty head, and

was like to die. Strong action was indicated, but what to do? Fertilizer, such as was used on the farm, was the answer, but the supply of fertilizer was definitely earmarked by superior order, for the farm alone. Private gardens, as far as these stern autocratic heads of camp were concerned, did not exist, and they cared not if gardenia bushes withered and blew away. But we cared, and determined to do something. I suppose we could have dug holes in the garden, and never gone near the buckets, but it was a biggish garden, and there were only three of us. We needed co-operation which was not forthcoming. We therefore steeled ourselves to crime. Late at night, when all good prisoners were supposed to be in bed, one of my medical friends and I, on a plea of urgent necessity, visited the latrine. We seized a bucket, well filled, and quietly carried it to our thirsty garden, which had been prepared to receive it, and there disposed of the contents. Now we were thieves. Crime seemed so simple, we wondered why we had ever tried to live honestly. But the bucket had to be returned. While my co-conspirator "filled in" in the garden, I carried the bucket back. And then, I shudder to think of it, I was caught. On the way back, I met one of the prisoners returning to his quarters. He said, "Good evening, sir," and saluted. Can anyone tell me the procedure in a case like this? How does a senior officer return a salute, while carrying a latrine bucket?

The veterans of any campaign, when they meet, love to swap stories of their exploits in the field. The veterans of Hong Kong have the same bad habits as the rest, but our reminiscences are largely bounded by barbed wire, and are chiefly of interest to our own little group. The stories I have told you today are the type of thing we tell among ourselves. If outsiders are interested to hear them, we can tell such tales indefinitely, so long as we excite only interest. But I for one, if ever I feel that I have excited pity, will become dumb, and spin my tales no more.

The bad things are forgotten, the good things are remembered, which is as it should be.

The real solution for the cancer problem does not lie in its surgical and radiation treatment. It lies rather in discovering the fundamental principle or principles wherein a healthy cell escapes its normal growth restraints and disregards its function as a serviceable unit of an organized whole.—*A. J. P. H.*, **37**: 1296, 1947.

MEDICO-LEGAL

The Work of the Canadian Medical Protective Association*

Admission of Liability

Several cases were referred to the Council of the Association during the past year where *ex gratia* payments have been made or liability admitted by doctors for mistaken diagnosis or treatment. In such cases there is nothing to prevent a claimant publishing to others the news that the doctor has made such payments and giving them his version of the facts. Too much stress cannot be laid on the importance of reporting any claim to the Council of the Association *before* any financial arrangement is entered into or admission made by doctor to patient.

In one case which occurred during the past year, a doctor undertook to pay a dentist's account for replacing certain front teeth which had been displaced by the slipping of a mouth gag during the performance of a tonsillectomy. Some time later the doctor was asked to pay an account totalling $1,500.00 which had been received from the dentist. Only then did the doctor concerned report the incident to the Association. Had the doctor concerned reported the matter immediately after its occurrence, he would have been advised what procedure to follow in view of the facts and the law. Advice should be obtained from the Association at the earliest possible stage before any commitment is made or action taken. This will benefit the practitioner who should always remember that if he exercises reasonable care and skill he cannot be held negligent even though some person of greater skill and knowledge might have prescribed a different treatment or have acted in a different way.

Consent to Operation

During the past year a surgeon was successfully defended in an action for assault and battery arising out of the sterilization of a wife following an abdominal operation. The wife had requested such an operation during a visit to her physician but the husband was never consulted before the operation took place. A jury found as a fact that the sterilization had been performed with the consent of the wife. It also found that the husband by his course of conduct before the operation had given a tacit or implied consent thereto.

Every human being of adult years and sound mind has a right to determine what shall be done with his own body; and a surgeon who performs an operation without his patient's consent, commits an assault, for which he is liable in damages. This is true except in cases of

* Extracted from the annual report by the General Counsel for the C.M.P.A.

emergency where the patient is unconscious and where it is necessary to operate before consent can be obtained. Reasonable latitude must, however, be allowed the physician or surgeon in a particular case; and the Courts will not lay down any rule which would unreasonably interfere with the exercise of his discretion or prevent him from taking such measures as his judgment dictated for the welfare of a patient in case of emergency. For instance if, in the course of an operation to which the patient consented, the physician or surgeon should discover a condition not anticipated before the operation was begun and which, if not remedied, would endanger the life or health of the patient he would be justified in performing an operation to remove or overcome the dangerous condition though no express consent was obtained or given.

It is most important that a written consent should be obtained from both husband and wife before an operation, where a physician or surgeon decides that it is in the interests of the patient's life or health to sterilize the patient.

X-RAYS

One of the commonest grounds for legal action against a medical practitioner is alleged failure to reduce a fracture properly and, particularly, failure to arrange for a check x-ray examination. If a practitioner relies on fluoroscopic examination to confirm reduction, but fails to take an x-ray plate which can be produced subsequently in court if need be to substantiate his statement that there was in fact a good result, then the attending physician may be in a difficult position if the patient or a relative institutes an action for negligence. In cases where an x-ray examination is refused, the patient should be asked to sign a statement indicating his refusal. It should be remembered that the possession of precise and contemporary case notes are sound evidence of a physician's conscientious attention to a patient.

MEDICAL SOCIETIES

Defence Medical Association of Canada

The Military District No. 4 Branch of the Defence Medical Association of Canada held their annual meeting in Montreal on Friday, October 3, 1947.

Officers elected for the coming year were: *President*—Brigadier H. M. Elder; *Vice-president*—Brigadier C. B. Thompson; *Secretary*—Colonel C. U. Letourneau; *Treasurer*—S/Cmmdr. H. S. Morton; *Members*—W/Cmmdr. T. E. Kirk and S/Cmmdr. A. Ross.

A vote of thanks was tendered to the out-going executive for their efforts during the past year. Major-General C. P. Fenwick, out-going President, informed the meeting that it was very gratifying to note that most of the recommendations passed at the Annual Meeting of the D.M.A. in November, 1946, had been implemented and that the new policies of the Medical Services were progressing favourably. Reports of the activities of the D.M.A. were read by Colonel J. P. Laplante, General Secretary. Report of activities of Defence Association were read by Brigadier H. M. Elder.

Resolutions passed at the meeting included the approving of Ross House as suitable quarters for R.C.A.M.C. units in Montreal, and the implementing at the earliest opportunity of the amalgamation of the Medical Services of the 3 Armed Forces. The meeting adjourned, to be followed by a social hour in the Officers' Mess.

N.B. Branch Defence Medical Association

The annual meeting of the N.B. Branch Defence Medical Association of Canada was held at Lancaster D.V.A. Hospital October 21. Dr. A. B. Walter was chairman. Election of officers resulted in the following slate: *President*—Dr. D. F. W. Porter of Fredericton; *Vice-president*—Dr. R. A. H. MacKeen of Rothesay; *Secretary Treasurer*—Dr. J. A. Melanson of Fredericton. Dr. D. F. W. Porter was elected delegate to the annual medical meeting of the association at Ottawa.

New Brunswick Medical Society

The Sixty-seventh Annual Meeting of the N.B. Medical Society was held in Moncton, September 30 and October 1, under the general chairmanship of Dr. W. S. Fitzpatrick, President of the Society. From the President's reception until the last visitor left town, the Moncton Medical Society, acting as hosts to the profession of the province, gave an example of that hospitality for which the "Hub City" is famous. The scientific program speaks for itself and the business sessions saw the new problems of the day carefully discussed and the general business of the Society put in order for another year.

Dr. F. G. McGuinness, President of the C.M.A. and Dr. A. D. Kelly, Assistant Secretary of C.M.A. were honoured guests and actively took part in our deliberations and discussions. Dr. J. P. McInerney, of Saint John won the annual Golf Award of the VanWart Challenge Cup. It was decided to negotiate for a new schedule of W.C.B. fees along the line laid down in the D.V.A. Schedule.

An informative discussion of pre-paid medical care was very general, guided by Dr. Geo. Skinner and Dr. Arthur VanWart and a special committee was appointed to further our efforts along this new departure in Canadian Medicine.

Possible amendments to the Medical Act were planned to aid in discipline of illegal practitioners.

The election of officers and committees was completed as follows: *President*—Dr. D. A. Thompson, Bathurst; *1st Vice-president*—Dr. G. E. Chalmers, Fredericton; *2nd Vice-president*—Dr. W. W. Fleck, Dalhousie; *Treasurer*—Dr. A. L. Donovan, Saint John; *Secretary*—Dr. F. C. Jennings, Saint John. *Executive Committee*—Dr. J. J. McPherson, Campbellton; Dr. E. O. Thomas, St. Stephen; Dr. R. D. Roach, Moncton; Dr. Geo. Skinner, Saint John; Dr. M. Veniot, Bathurst; Dr. D. Albert, Edmundston; Dr. R. B. MacKenzie, Newcastle. *Members of the N.B. Medical Council*—Dr. W. L. Earle; Dr. J. R. Nugent; Dr. H. E. Britton, Dr. A. S. Kirkland, and Dr. P. C. Laporte. Dr. J. M. Barry is the Registrar of the N.B. Medical Council. *W.C.B. Buffer Committee*—Dr. G. F. Skinner, chairman; Dr. J. P. McInerney; Dr. Geo. White. *Cancer Committee*—Dr. J. R. Nugent, chairman; The President, the Secretary and the Registrar are Dr. J. S. Hynes and Dr. H. R. Ripley. *Golf Committee*—Dr. A. F. VanWart, Dr. V. D. Davidson, and Dr. J. P. McInerney. *Additional Members of C.M.A. Council*—Dr. A. F. VanWart; Dr. Geo. White; Dr. M. L. Rice; Dr. W. Ross Wright; Dr. Geo. Skinner; Dr. H. E. Britton and Dr. E. Dumant. *C.M.A. Nominating Committee*—Dr. Geo. White; alternative, Dr. H. Everett. *C.M.A. Executive Committee*—Dr. A. VanWart; alternative, Dr. Geo. Skinner: *Divisional Representative C.M.A. Editorial Board*—Dr. A. S.

Kirkland. *Representative to N.B. Tuberculosis Association*—Dr. Norman Skinner. *D.V.A. Medical Advisory Committee*—Dr. G. B. Peat, chairman; Dr. J. R. Nugent; Dr. F. C. Cheesman; Dr. F. R. Connell and Dr. A. L. Donovan. *Hospital Planning Committee*—Dr. C. L. Grass. *Prepaid Hospital and Medical Care Committee*—Dr. VanWart; Dr. W. S. Fitzpatrick and Dr. Alec. McClennan.

The provincial cancer plan was the subject of much discussion, and the new set up of cancer diagnostic centres in the established hospitals of the province by the provincial department of health was commended and approved by formal motion of the society.

Regina and District Medical Society

A well attended meeting of the Regina and District Medical Society was held on Friday, October 17, at the Hotel Saskatchewan. Dr. D. C. MacRae, the new President, was chairman.

The program was taken over by Dr. A. W. Thomson, who gave an excellent historical account of medical practice in Regina nearly fifty years ago. Dr. E. A. McCusker reported to the district on the activities of the College of Physicians and Surgeons. He is their representative on the Council and desired to acquaint them with the progress of their association work. The third speaker, Dr. D. G. McKerracher, Commission of Mental Services, gave a very complete and clear outline of the plans in Saskatchewan for the care and management of the mental defective and sub-normal child.

Saskatoon and District Medical Society

A dinner meeting of the Saskatoon and District Medical Society was held on Tuesday, September 30. An excellent attendance was noted, including several men from the surrounding rural areas. The business of the evening consisted of a full discussion and reporting by authorized committees of association business. Dr. J. F. C. Anderson reported up to date on the business of the College of Physicians and Surgeons. It is hoped in the near future to have a mixed dinner. The opinion was expressed that the Doctors' wives are just as interested in professional affairs as the Doctors themselves.

Swift Current and District Medical Society

A meeting of the Swift Current and District Medical Society was held at Gull Lake on October 10, 1947. Dr. Matheson, President, was in the chair and the district was well represented, there being about twenty doctors present. Dr. Dunn, regional radiologist, gave an interesting discussion on x-ray films and showed some outstanding pictures. When he had finished a vote of thanks was tendered to him and a sincere expression of regret made to him because of his imminent departure. The business side of the meeting involved a discussion in financial arrangements with the Health Region and a committee of three was instructed to interview the regional executive to ascertain the exact position. Dr. N. E. Dunn is relinquishing his position as radiologist of Swift Current Health Region No. 1 and is moving to take the position as radiologist in Moose Jaw.

La société médicale des hôpitaux universitaires de Québec

Société médicale des hôpitaux universitaires de Québec, le 3 octobre, 1947.

HYPERTENSION ARTÉRIELLE ET ÉLECTRO-CHOC. — Sylvio Caron, C.-A. Martin et L.-H. Lemieux.

De l'étude de trente-huit malades mentaux hypertendus soumis à l'électro-choc, il ressort que: (1) L'hypertension artérielle rencontrée chez le malade psychiâtrique, est le plus souvent d'origine psychogène. Elle accompagne surtout les états d'anxiété marquée. (2) Elle s'abaisse le plus souvent au cours de la convulsivothérapie à peu près parallèlement à l'amélioration de la maladie mentale. (3) Les malaises subjectifs qui lui font cortège sont surtout d'ordre psycho-névrotique et disparaissent en même temps que guérit l'état mental. (4) Nos hypertendus au-dessus de 200 sont toujours de grands anxieux, avec des complexes affectifs solidement ancrés. Ils nécessitent un plus grand nombre d'électro-chocs. Quand il y a guérison, le pronostic reste réservé au point de vue psychique. (5) L'hypertension artérielle, isolée même si elle est marquée, n'est pas une contre-indication à l'électrochoc. Il faut d'abord s'assurer de l'intégrité du système cardio-vasculaire en faisant tous les examens et surtout celui du fond de l'œil. Ces malades doivent être surveillés plus étroitement en cours de traitement. (6) Enfin l'électro-choc n'est pas un traitement spécifique de l'hypertension. Il est cependant possible que le jour où l'on aura réalisé la convulsivothérapie sans convulsion, on aura du même coup découvert un traitement plus logique de l'hypertension artérielle d'origine neurogène.

MALFORMATIONS CONGÉNITALES (Micromélie-Macromélie). —Geo.-H. Larue, P. Michel et G. Paradis.

Présentation de 2 cas de malformations congénitales. Le premier une micromélie: Arrêt de développement osseux des 4 membres; os courts, déformés par de nombreuses courbures donnant au patient l'aspect d'un nain. Dystrophie osseuse rappelant la dysplasie pèriostale de Durante, mais s'en différenciant par les caractères radiologiques. Un deuxième cas, macromélie: Enfant de 10 ans avec le membre inférieure droit du volume de celui d'un adulte. Malformation datant de la naissance. Pied droit énorme. Début d'hyperplasie semblable au pied gauche.

MALADIE DE RECKLINGHAUSEN.—M. Samson et Laurent Patry.

Deux observations de maladie de Recklinghausen, dont l'une a été une manifestation aigue de la maladie et dont l'évolution a paru être aggravée par l'accouchement. Découverte à l'autopsie d'une grosse tumeur de la surrénale gauche, avec néoplasie volumineuse envahissant les lobes pariétaux et occipitaux et la partie postérieure des noyaux gris de l'hémisphère droit (gliôme). Dans la deuxième observation, la maladie a évolué par poussées d'une façon très lente et progressive, on a pu retracer le caractère héréditaire, congénital et familial de la maladie. La symptomatologie est au complet avec association de lésions osseuses contrôlées par la radiographie.

DEMENCE ARTERIO-SCLEREUSE.—S. Caron.

Le docteur Caron profite de l'occasion de cette présentation pour rappeler aux membres de la Société les différents aspects cliniques de la cérébro-sclérose, c'est-à-dire le syndrome pseudo-bulbaire, la paraplégie, la neurasthénie, le parkinsonisme, l'épilepsie et l'état démentiel; puis il fait remarquer l'âge précoce du sujet, 59 ans, et la céphalée frontale droite qui apparait à chaque jour depuis trois mois et qui s'accompagne de manifestations pathologiques rappelant celles de l'épilepsie larvée.

Il donne les raisons d'éliminer la maladie de Pick et d'Alzheimer, ainsi que la tumeur cérébrale. L'encéphalographie qu'il a pratiquée lui permet de rejeter l'hypothèse d'une tumeur cérébrale.

FATIGUE ET FOIE DE VEAU.—C.-A. Martin.

Le foie de veau a été employé dans le traitement de diverses affections, sans qu'on sache bien encore tout ce qu'il contient. Il n'a une action spécifique que dans les anémies macrocytaires. Il ne vaut absolument rien contre la fatigue, qui le plus souvent est un symptôme d'origine mentale. Il peut même empirer la maladie en causant de l'angoisse. L'auteur s'élève contre l'emploi abusif qui en est fait.

SPECIAL CORRESPONDENCE

The London Letter

(From our own correspondent)

DECLINE IN POLIOMYELITIS

The steady decline in notifications of poliomyelitis suggests that the epidemic is now spending itself. The latest figures, 338 during the week ending October 11, are the lowest recorded for eleven weeks. It has just been made known that two large-scale inquiries concerning the disease are in progress. One, under the ægis of the Ministry of Health, aims at securing more accurate estimates of the fatality and severity of the disease in the present epidemic. The other is being carried out by the Public Health Laboratory Service and is concerned with the epidemiology of the condition. Throughout the epidemic every effort has been made to collect all available data, and it is hoped that when these are duly correlated a useful addition will be made to the already large mass of factual information concerning this perplexing disease.

MATERNAL NUTRITION AND FETAL MORTALITY

One of the most striking features of the war years has been the marked decline in the still-birth rate. In England and Wales, for instance, where for many years before the war there had been little change in the rate, it fell from 38 to 28 between 1939 and 1945. In Scotland the fall during the same period was from 42 to 33. Professor Dugald Baird of Aberdeen has now published a report suggesting that this fall is due largely to improved diet during pregnancy. After showing that the health, physique and diet of the higher income-groups is much better than those of the lower income-groups, he correlates this with the "reproduction performance" by showing that the still-birth rate in the lower income-groups is between two and three times that in the higher groups. It is in the former groups that the greatest fall in the still-birth rate occurred between 1939 and 1945.

To explain the fall in the still-birth rate which also occurred in the higher income-groups in which the pre-war purchasing power was sufficient to ensure an adequate diet, it is suggested that this may have been due to the publicity given during the war to the importance of sound nutrition. Other factors, such as the standard of medical supervision during pregnancy and labour, undoubtedly also play a part, but a good case has been made out for the outstanding importance of a satisfactory state of nutrition in ensuring the birth of a viable healthy child. It is also a striking confirmation of the pioneer work of the Toronto workers.

THE BATTLE OF THE BOOKS

Of all the recent bans and restrictions imposed by the Government none is more reprehensible than that forbidding the import of foreign books except under individual licence. So far as the medical and scientific professions are concerned it is American books which are mainly involved, and it is difficult to exaggerate the hardship that the loss of such books will impose upon teachers and research workers in these subjects. Had the ban been compensated for by some loosening of the stranglehold which is slowly strangling British publishers, the hardship would not have been so great, although the fundamental objection would not have been met. Apparently, however, in these materialistic days of rationalization the things of the spirit and of the mind are of minor consideration, and so long as dollars are saved the resulting intellectual impoverishment is of secondary importance. This is a tendency to which there is a real danger of our being acclimatized, and it is only to be hoped that the rugged independence of our men of science and of the professions will successfully overcome this latest attack on the free interchange of ideas between nations.

FOOD AND DRINK INFECTIONS

The curious backwardness of our legislation controlling the handling of foodstuffs has been re-emphasized at a recent conference convened by the Central Council of Health Education. Among some of the more interesting facts brought out at the conference were that 1,500 to 2,000 people in this country die annually from tuberculosis caused by infected milk, and that in 1944 more than 200 people died of enteric fever and dysentery, and 3,700 children under the age of two years died of diarrhœa and enteritis. There has been a seven-fold increase in the number of food-poisoning outbreaks since before the war, there now being an annual incidence of 500. An instance was quoted of how such attacks occur: a large outbreak was traced to a cook with sores on his hands infecting a potato salad which was eaten by hundreds of people attending an agricultural show. Perhaps uncontrollable by any legislation was the case of the gentleman selling milk shakes at the roadside who, when he ran out of water, used water from a neighbouring ditch: twelve of his customers developed typhoid fever! That improvements can be introduced by voluntary methods was shown by one speaker who described how in Scotland food handlers serving holiday camps had been persuaded, with only a few exceptions, to submit to medical examination. This examination, which included bacteriological examination of the excreta, revealed the presence of several carriers.

WILLIAM A. R. THOMSON

London, November, 1947.

ABSTRACTS FROM CURRENT LITERATURE

Medicine

The Antihistaminic Drugs. Waldbott, G. L.: *J. Am. M. Ass.*, **135:** 207, 1947.

Since the indiscriminate use of the new antihistaminic drugs has become so widespread, and may have harmful results, a critical inventory of their value is offered. The mechanism of allergy is briefly described and a historical and descriptive survey of the various remedies which have been employed for its control is given. These include the attempt to eliminate the antigen from the environment and ingesta; attempts to produce protective antibody by hyposensitization; the use of epinephrin and ephedrin to contract the capillaries and relieve smooth muscle spasm, as by aminophyllin in asthma; and attempts to counteract the effect of histamine. Histaminase (torantil) is successful *in vitro*, but has not proved to be of clinical value, and an attempt to produce immunity to histamine by a histamine-protein compound (hapamin) has also been unsuccessful. Many of the compounds synthesized in Europe and America, following the demonstration by Fourneau and Bovet in 1933 that certain phenolic ethers counteracted the action of histamine, have proved too toxic for clinical use. The most promising ones have been marketed under the following names: "antistin", "neoantergan", "pyribenzamine hydrochloride" and "benadryl hydrochloride". They counteract most of the physiologic effects of histamine, but do not chemically neutralize histamine nor prevent its production in the body. They are believed to compete with the histamine in its affinity for cells. Clinically their ability to inhibit whealing and to dry up mucous secretion is outstanding. Hence their conspicuous success in urticaria, where their efficiency is rated at 85 to 95%, and hay fever 70 to 80%. In descending order of efficiency the author gives after these, vasomotor rhinitis, asthma, pruritus of eczema and contact dermatitis and migraine. The untoward effects are divided into side effect (dizziness and drowsiness in 20 to 50%, dryness in mouth, headache, nausea and vomiting, muscular twitching, paresthesia and dilated pupils); acute (one

case only reported) and chronic (granulocytopenia?) toxicity; and allergic symptoms (asthma, cough, dermatitis and urticaria, the last two being extremely infrequent). Prolonged administration has not so far resulted in any demonstrable organic damage either in human beings and in animals, and there is so far no indication of cumulative action or addiction. Their purely palliative effect must be recognized, but in this they are equal or superior to such agents as epinephrin, ephedrin or aminophyllin. D. E. H. CLEVELAND

Aspirin Allergy. Friedlander, S. and Feinberg, S. M.: *Ann. Int. Med.*, **26**: 734, 1947.

According to the authors allergic reactions to aspirin are commonly encountered. Asthma and urticaria are the most common manifestations, and when they occur are extremely acute in nature and difficult to relieve. The frequent association of aspirin allergy with cases of severe chronic asthma having a poor prognosis, prompted a review of findings in 45 patients. A high incidence of familial allergy, emphysema, nasal allergy with polyposis, eosinophilia, and frequent absence of positive skin reactions to specific agents were found. Attention to specific factors where identified, resulted in some improvement. In the majority, where etiological factors could not be determined, management based on control of associated infectious elements proved disappointing. Non-specific methods of treatment were of limited value.

Since aspirin in these patients produces acute episodes of asthma, yet fails to give positive skin reactions, it is suggested that repeated exposure to other substances of a chemical nature, also incapable of giving positive skin tests may be responsible for the chronic asthma encountered in these individuals. Such substances could be present in the diet or environment. Allergy to aspirin may be an indicator that the individual has acquired the mechanism to react to simple chemical compounds. Such a mechanism could explain the many cases of allergic disease in which a specific protein allergen cannot be identified. S. R. TOWNSEND

Four Important Congenital Conditions Causing Cyanosis to be Differentiated from the Tetralogy of Fallot. Alexander, F. and White, P. D.: *Ann. Int. Med.*, **27**: 64, 1947.

Cases have been presented to illustrate four noteworthy congenital cardiac conditions causing cyanosis which must be differentiated from the more common tetralogy of Fallot. These four conditions are tricuspid atresia, Eisenmenger's complex, transposition of the great vessels, and a single ventricle.

Emphasis has been placed in this paper on the case of tricuspid atresia, and also on its clinical features, which render it a definitely diagnosable entity. The case herewith presented is that of a five and one-half month old boy in whom periodic attacks of cyanosis were noted by the mother, and who showed on physical examination a generalized slate-blue colour, a grade 4 systolic murmur in the second and third interspaces, just to the left of the midsternal line, and also a palpable liver. By electrocardiogram he presented definite left axis deviation, by x-ray an enlarged heart with no evidence of pulmonary engorgement.

A case of probable Eisenmenger's complex has been described and discussed briefly. This was a five-year old boy who complained of intermittent attacks of shortness of breath and cyanosis, and on physical examination demonstrated a rather persistent cyanosis, an enlarged heart, with a grade 2 systolic murmur, and a barely palpable liver. X-ray of the chest showed pulmonary congestion. The electrocardiogram showed right axis deviation.

Description of a case of transposition of the great vessels with explanatory notes referable to its embryology and clinical findings was presented. The patient was a seventeen-year old boy who was said to have been short of breath and cyanotic for the greater part of his life and to have been hoarse and to have manifested some degree of epistaxis only within recent months. Physical examination revealed chiefly a deep cyanosis, clubbing of the digits and toes, an enlarged heart, without murmurs or evidences of pulmonary congestion. X-ray confirmed the heart size and suggested the possibility of transposition of the great vessels. An electrocardiogram demonstrated right axis deviation.

Lastly, a case of a single ventricle was described. The child, a one month old boy, was noted to have periodic episodes of cyanosis, and also some difficulty in breathing. By physical examination he was moderately dyspnœic and cyanotic, with an enlarged heart, a grade 3 systolic murmur at the apex, and a palpable liver. Right axis deviation was prominent in the electrocardiogram. S. R. TOWNSEND

Early Amyloid Nephrosis in Complicated Traumas of the Bone Sustained During War. Rieselman, S. D.: *Arch. Int. Med.*, **80**: 165, 1947.

That there can be a connection between osseous and renal changes has been known for many years. Renal dwarfism, renal rickets are terms indicating a search for some way of labelling such cases. Renal insufficiency and some kind of disturbance in mineral metabolism, it was thought, both played a part in producing the clinical pictures, parathyroid hyperfunction and enlargement being one of the findings, probably caused by retention of phosphate. Rusakov felt that every case of long standing renal insufficiency entails hyperplasia of the parathyroids.

Formation of calculi in kidneys is often observed in various neoplasms and inflammatory processes in bones causing extensive destruction of bone and invasion of the blood stream by the products of altered calcium metabolism lead to the formation of amyloid casts in the kidneys. If this process advances far enough there will be occlusion of the nephrons with renal failure. Formerly thought to be a disease of very slow development, recent opinion tends to the belief that amyloid nephrosis can develop very rapidly. Early recognition, again, can lead to successful treatment. For instance œdema is found early while albuminuria may not be constantly present. Another symptom of amyloid nephrosis is diarrhœa when intestinal amyloidosis is present. Several cases are reported where serious injuries such as compound fractures of heavy bones were later complicated by the development of general œdema and other evidence of amyloid nephrosis; sometimes not diagnosed until autopsy. The interval between injury and onset of œdema varied between 2 to 3 weeks and 2 to 3 months. P. M. MACDONNELL

Idiosyncrasy to Drugs. Dragstedt, C. A.: *J. Am. M. Ass.*, **135**: 133, 1947.

Early writers used the term idiosyncrasy to describe an abnormal response to drugs, which could be either quantitative or qualitative. A characteristic and wide normal variability in the quantitative response is still recognized. Where data are available figures can be established expressing the normal range of quantitative variability in the case of a given drug, and susceptibility at or beyond the extremes established could be called tolerant on one hand or hypersensitive on the other. The term idiosyncrasy has thus come to have the narrower meaning of quantitative abnormality in the response to drugs, signifying such effects as urticaria, neuritis, jaundice and leukopenia produced in some persons but not in the majority, irrespective of the dose. Experience has taught that some drugs such as acetylsalicylic acid, aminopyrine, organic arsenicals, sulfonamides, thiouracil and quinine produce idiosyncratic reactions more frequently than alcohol, amphetamine, cascara, chloral hydrate and digitalis, but it is impossible to make prophetic generalizations. Certain drugs, by forming antigenic conjugates with body proteins produce their effects by an immunologic reaction, which is called allergic. Some of the toxic reactions produced by a drug are not idiosyncratic at all. Examples are the suppression of urine produced by excretion and pre-

cipitation in it of sulfonamides, or the prothrombinæmia produced by large doses of salicylates. Some reactions to drugs meet the criteria characteristic of allergic reactions. Such are the reactions to acetylsalicylic acid, and a number of those due to sulfonamides and penicillin. Again in other types of idiosyncrasy the weight of evidence is against the allergic nature of the reaction. Examples are jaundice with or without acute yellow atrophy of the liver due to various drugs, and probably the granulocytopenia produced by thiouracil and some organic arsenicals. It is concluded that a variety of drug idiosyncrasies may depend upon nonallergic factors and probably some drugs may produce both allergic and nonallergic types of toxic reaction at the same time.

Dragstedt suggests as provisional criteria (1) that an allergic reaction is indicated when the pattern of the toxic reaction is that of allergic reactions produced by antigenic agents; such reactions as urticaria, dermatitis and asthma being allergic; reactions characterized by jaundice, acute yellow atrophy and optic atrophy being nonallergic, while granulocytopenia, anæmia, thrombocytopenia and polyneuritis may well be one or the other; (2) An allergic basis is indicated by a prior sensitizing administration of the drug; a nonallergic basis when long-continued or large doses have been followed. (3) An allergic basis is suggested when reactions are alleviated by epinephrin, benadryl and similar agents; a nonallergic basis when alleviated follows exhibition of ascorbic acid, folic acid or thiamine. Chemists, who have already synthesized efficient sulfonamide compounds and propylthiouracil to replace sulfapyridine and thiouracil, are looked to for other products which will produce the effects of drugs now used, but with fewer untoward effects.

D. E. H. CLEVELAND

The Use of Dicoumarol as an Anticoagulant. Allen, E. V. *et al.*: *Ann. Int. Med.*, **27**: 371, 1947.

The expert use of the anticoagulants, heparin and dicoumarol, has improved tremendously the outlook for patients who have acute vascular thrombosis. An overall consideration of 1,513 postoperative patients treated with anticoagulants indicates that the following results were achieved: 85 patients survived who would have been expected to die from pulmonary embolism; 250 patients were spared venous thrombosis or non-fatal pulmonary embolism. In 506 additional postoperative cases in which dicoumarol was used prophylactically, venous thrombosis occurred in but two instances; there was no pulmonary embolism.

A consideration of 288 medical patients indicates that fatal pulmonary embolism was prevented by anticoagulants. Non-fatal pulmonary embolism and venous thrombosis occurred very infrequently. A study of 50 cases of acute myocardial infarction indicates substantial reduction in the incidence of further myocardial infarction and in arterial embolism and venous thrombosis. Survival of the extremity occurred in 91% of cases of arterial embolism, and in 81% of cases of arterial thrombosis, if treatment with anticoagulants was begun early and supplemented by other treatment.

In general, the use of anticoagulants constitutes the greatest contribution to the successful treatment and prevention of intravascular thrombosis and embolism.

S. R. TOWNSEND

A Study of Mitral Stenosis in Patients Who Survived the Age of Fifty. Baker, L. A. and Musgrave, D.: *Ann. Int. Med.*, **26**: 901, 1947.

A study of the clinical course and findings in 105 patients with isolated mitral stenosis who survived the age of 50 has been made by the authors. The high incidence of a negative history of rheumatic fever is thought to be due to these patients having suffered from atypical "rheumatic type" infections during which cardiac damage was done. The information indicates that the advanced age of these patients is associated with an unusually long course of rheumatic heart disease. This long course can be explained by a fundamental difference in the pathogenesis of the rheumatic process within the heart in contrast to that occurring in younger persons with a shorter duration. A relatively slight involvement of the heart, limited for the most part to the endocardium and mitral valve would allow the development of mitral stenosis with the myocardium being spared. Under these circumstances, the heart is able to maintain compensation over a period of many years, and in many instances to tolerate active physical exertion.

The great frequency of symptoms and physical findings of non-tuberculous pulmonary disease was striking in these patients. It is the opinion of the authors that the longer the patients with mitral stenosis live, the greater will be the incidence of chronic pulmonary disease. The incidence of auricular fibrillation was high and is believed to be related to the duration of auricular strain. Embolic manifestations were frequent, occurring in the absence of, as well as with auricular fibrillation. Hypertension was not as common in this group of patients as has been reported by other writers. It apparently was a factor in causing a marked degree of cardiac enlargement in many instances. It is not believed that the longevity of these patients is dependent upon the presence of hypertension as has been suggested.

Factors which are commonly present in these older patients and add to the difficulty in diagnosis are discussed. Certain clinical observations which should suggest the possibility of mitral stenosis are outlined. The majority of deaths in this series were due to the cardiovascular disease present. In approximately two-thirds of the fatal cases death was from congestive failure.

S. R. TOWNSEND

Surgery

Needle Biopsy in the Clinical Diagnosis of Tumours. Ellis, F.: *Brit. J. Surg.*, **34**: 240, 1947.

Based on an experience with 280 aspiration biopsies, the advantages and risks of needling or drilling for a section of a tumour are described. Useful information was obtained in 60% of the cases undertaken. Needle biopsies are indicated (1) when the tumour lies deep to normal skin or other tissues; (2) when the tumour may be infected; (3) when a major operation would otherwise be necessary yet unlikely to allow successful extirpation of the tumour; (4) when usual methods of obtaining histological evidence are contraindicated; (5) when Hodgkin's disease is suspected and cancer may be ruled out. Clinical evidence unsupported by histological findings is misleading and doubtful in evaluating five-year cures by radiotherapy.

Methods of obtaining aspiration biopsies from bones, glands and various organs are described, and details of the methods of embedding and studying the cores of tissue obtained are described. Types of tumours thus diagnosed are listed and illustrated ranging through tuberculosis, chondromas, carcinomas, mixed tumours, lymphomas, melanomas, Ewing's tumour, sarcomas, seminoma, Hodgkin's, osteoclastoma. The technique and apparatus used in making the drill biopsy are described and show advantages in greater control, larger pieces of tissue, and easier operation especially when a motor drill is available. The drilling needles were designed especially for the author.

In this series only two immediate and two late complications occurred. The pathologists were converted from early doubt to enthusiastic interest. It is believed that initiation of rapid spread of a malignant tumour is much less likely to happen in this type of biopsy.

BURNS PLEWES

Tumours of the Carotid Body. Labey, F. H. and Warren, K. W.: *Surg., Gyn. & Obst.*, **85**: 281, 1947.

Carotid body tumours have been resected since 1880 and may attain a size up to 190 grams. Two types of cell patterns have been described: alveolar and peritheliomatous, and malignant changes have been found in 15 to 20%, though distant metastases are rarely, if ever, seen. Often the tumour so surrounds and fuses

with the external, internal and common carotid arteries that these vessels must be ligated; a procedure accompanied by a forbidding morbidity and mortality. No one has reported a large series of cases, there being only 300 in the literature. This article reviews 18 carotid body tumours.

Lateral tumours of the neck that are single, discrete and movable, consist of branchial cysts, carotid body tumours, single large aberrant thyroid, and neurofibromas, in that order of frequency. Branchial cysts are superficial in the lower neck; carotid body tumours are deep and enlarge upward under the angle of the jaw.

Before undertaking an operation for the removal of a tumour which may be of the carotid body, the surgeon must know that cerebral symptoms do or do not occur on complete common carotid compression for 10 minutes three times a day. Even if no symptoms occur, disaster may follow carotid ligation, so the test is not dependable. Though the ideal treatment is complete surgical extirpation, the risk of ligature of the carotid is such as to justify not employing this procedure. In 75% the tumour is the cause of no symptoms and in the rest the symptoms are trivial. The tumour usually grows very slowly. When malignancy occurs, it is of low grade. Local lymph nodes may be invaded but distant metastases have never been verified.

Of the 18 patients, 17 complained of a swelling in the neck, 5 had pain in the neck or face, 3 had hoarseness, 1 had dysphagia. In 3 the tumour was bilateral. There was no sex difference. In 2 the lesion bulged into the pharynx. An accurate diagnosis was made in 50%. Punch biopsy seems dangerous. In 5 cases the tumour was removed without carotid ligation. In 6, the common, external and internal carotids were tied. In 5, removal of the tumour was not carried out at operation rather than ligate the vessels. One patient out of the group not ligated died of carotid thrombosis. Thirty per cent of the patients with ligation died. It is doubtful that irradiation by x-rays or radium is beneficial. It is the belief of the authors that only if biopsy shows a malignant tumour, should such a lesion, involving the carotid arteries, be removed. If this is done, the internal jugular should also be ligated, and the patient nursed head down, in an oxygen tent for some time.

Since aberrant thyroid tumours are frequently malignant and are hard to differentiate from carotid body tumours, and since branchial cysts are easily taken out, and some carotid body tumours can be removed without ligation, all laterally located, discrete, and movable neck tumours should be explored. BURNS PLEWES

Obstetrics and Gynæcology

The Antenatal and Postnatal Care of the Erythroblastotic Infant. Bowley, C. C.: *J. Obst. & Gyn. Brit. Emp.*, **54:** 489, 1947.

Lesions of the central nervous system following erythroblastosis have already been briefly referred to. It seems probable that these lesions are due, not to the jaundice *per se*, but to a prolonged cerebral anæmia. There is the parallel case of the cretin. A cretin showing signs of mental deficiency within a week or two of birth will never recover with treatment as well as the case where mental signs do not become apparent until 3 or 4 months of age. This suggests that the neurons are more sensitive to slight changes during the few weeks following birth than later on. In support of this view the cranial nuclei of one 3 days old erythroblastotic baby, which died a few minutes after arrival at the hospital, showed deep yellow staining, but histological examination did not demonstrate any abnormality of the nervous system. Also infants jaundiced from other causes are not subject to such nervous sequelæ. This theory is a reason for transfusion if the hæmoglobin level drops below about 90% in the first week, or 80% later on. Levels of 50 or 60% should certainly not be allowed to occur. Finally, breast feeding must be prevented to avoid antibody transfer. P. J. KEARNS

Calcium and Phosphorus Metabolism in Pregnancy: A Survey Under War and Post-War Conditions. Obermer, E.: *J. Obst. & Gyn. Brit. Emp.*, **54:** 432, 1947.

On the basis of the figures given in this and the two previous papers, which cover 227 forty-eight hour investigations, at different phases of pregnancy, the following tentative conclusions have been arrived at: (1) That the phosphorus intake of normal pregnant women on a generous all round diet is usually adequate. When the diet is insufficient owing to poverty or any other reason, and we suspect that the daily intake of phosphorus is less than 1.5 g. per day, a supplement of phosphorus should be given. (2) That the minimun safety levels of daily calcium intake are 2 g. of calcium element from the beginning of pregnancy to the end of the 7th month and 2.5 g. from the 8th month to term. These amounts cannot be arrived at without supplement, however generous and all-round the diet. A pregnant woman who consumes her statutory pint of milk per day (0.72 g. Ca.) together with her daily ration of cheese (0.08 g. Ca.) and a high average of "other foods" intake (0.30 g. Ca.) arrives at a total 24-hour intake of only 1.1 g. To ensure adequate reserves every pregnant woman should be given a supplement of calcium phosphate, containing at least the following amounts of calcium and phosphorus elements: 3rd to 5th months, Ca. 0.64 g. P. 0.282 g.; 6th to 8th months, Ca. 0.95 g. P. 0.423 g.; 8th to term, Ca. 1.26 g. P. 0.564 g. P. J. KEARNS

Congenital Aneurysm of the Circle of Willis Associated with Pregnancy. Rhoads, E. E.: *Am. J. Obst. & Gyn.*, **54:** 533, 1947.

Two cases of congenital aneurysms of the circle of Willis associated with pregnancy are presented. The seriousness of such vascular lesions has been shown by statistical reports: the prognosis should always be guarded. If a patient has a past history of hæmorrhage from the rupture of an intracranial blood vessel, future pregnancies must be prevented, preferably by sterilization. Pregnancy adds gravity to the prognosis because of the increased blood volume; the increased pressure of the uterus or renal vessels, and the increased strain during labour. Termination of pregnancy is justified in all cases of proved bleeding from intercranial aneurysms. ROSS MITCHELL

A Further Contribution to the Study of the Influence of Failure of Mullerian Duct Fusion on Pregnancy and Labour. Way, S.: *J. Obst. & Gyn. Brit. Emp.*, **54:** 469, 1947.

A further 19 cases of minor degrees of failure of fusion of the Mullerian ducts are described. The details of 78 pregnancies are given. The commonest complications observed were transverse lie (27%), abortion (26%), and retained placenta (15%). The management of these is discussed. A plea is made for the detection of these cases during the antenatal period and for their delivery in hospital. P. J. KEARNS

The Excretion of Drugs in Human Milk. Sapeika, N.: *J. Obst. & Gyn. Brit. Emp.*, **54:** 426, 1947.

One of the earliest reviews of this subject was made by Reed (1908) many of whose observations still hold good today. The fact that it is practically never necessary to remove the child from the breast when the mother is receiving drug therapy is borne out by the rarity of cases of untoward reactions reported in the literature. Another important point that still holds is that it is rarely if ever desirable to treat a child with drugs given to the mother. From the clinical and qualitative chemical evidence at that time Reed concluded that alcohol, opium, chloroform, ether, thyroid, and lead are to be used with care, and that atropine, antipyrine (phenazone), phenacetin, chloral, arsenic, bismuth, mercury, copper, castor oil, senna, quinine, bromides, iodides, salicylic acid, and copaiba, pass into the milk in small

quantities. An almost identical list is given by Stander (1941). In the account presented here the reports since 1908 have been studied. The vitamin content of human milk has not been considered. The author has made extensive study of drugs excreted through lactation and listed them. P. J. KEARNS

Further Observations on the Use of the Neutral Diet and Hydration in the Treatment of Toxæmias of Late Pregnancy. De Alvarez, R. R.: *Am. J. Obst. & Gyn.*, **54:** 445, 1947.

Toxæmias of pregnancy are treated according to a conservative regimen, consisting of hydration, neutral diet, ammonium chloride, bed rest and mild sedation. Conservative therapy of pregnancy toxæmia embodies the modern principles of water balance. When conservative methods fail to control pregnancy toxæmia, termination of pregnancy is effected by the most conservative means suited to the particular individual. The latent sequelæ following pregnancy toxæmia are minimized by such conservative therapy. The indications for Cæsarean section in toxæmia patients should be essentially the same as when no toxæmia exists. ROSS MITCHELL

The Relation Between the Results of ABO and Rh Tests and the Clinical Condition of 2,000 Mothers and Infants. Boorman, K. E. and Daley, D.: *J. Obst. & Gyn.*, **54:** 281, 1947.

Perhaps the most interesting finding arising from the investigation, and one which can only be discovered from the testing of a large unselected series, is the fact that anti-Rh agglutins can occur in the maternal serum associated with a normal Rh positive infant. In our series we found 11 such cases (4 of these infants showed a mild transient jaundice). Continued investigation will probably lead to a new treatment of the hæmolytic disease of the fetus.

The husbands of all Rh negative women after the first pregnancy should be tested in order to find out whether they are Rh positive and if so whether homozygous or heterozygous. All Rh negative women who have more than one pregnancy should have their sera tested for Rh. antibodies at intervals during pregnancy especially between the 34th week and term as it is during this period that these antibodies are most likely to be detectable. P. J. KEARNS

Sedation with Rectal Tri-Brom-Ethanol (Averton, Bromethol) in the Management of Eclampsia. Dewar, J. B. and Morris, W. I. C.: *J. Obst. & Gyn. Brit. Emp.*, **54:** 417, 1947.

We have come to have such great confidence in the effects of bromethol that we are now disposed to take a much more conservative view of the management of pre-eclampsia. We believe that in bromethol we have a sedative agent which enables us to preserve with conservative treatment even in the presence of urgent prodromal symptoms of eclampsia, since we can confidently expect to control any seizures which do actually develop. We realize that the severity of eclampsia is controlled by factors additional to the number of fits and that it is unlikely that these factors will be influenced at all favourably by bromethol, possibly the reverse. Nevertheless, this trust in our sedative agent has given us added confidence in the handling of these difficult cases.

In parenthesis, we think it permissible to refer to the fact that in many cases of severe pre-eclampsia we have used bromethol in the initial treatment and during labour, and that no case has developed fits when under its influence. We have also used bromethol from time to time as an analgesic in normal labour, but are not so favourably impressed with it in this field. Sedation has seemed to us more difficult to achieve than in the eclamptic. We do not wish to decry bromethol in this respect but in our hands it has proved erratic.

P. J. KEARNS

Neurology

Changes in Sensory Adaptation Time and after Sensation with Lesions of Parietal Lobe. Bender, M. B.: *Arch. Neurol. & Psychiat.*, **55:** 299, 1946.

The author describes a patient with a gunshot wound of the left posterior parietal and occipital lobes, showing disturbances in visual, cutaneous and proprioceptive senses on the right side of his body. These defects were elicited under special conditions. Whereas with single stimulation his vision appeared to be intact, simultaneous stimulation of his right and left fields of vision caused the image on his right to become extinct. This phenomenon of extinction was found in various forms, from fluctuation and blurring to complete invisibility of an image. A similar phenomenon was found for cutaneous perception. Besides this, the patient showed a reduction in the adaptation time for cutaneous and proprioceptive modalities. The latter was pronounced enough to produce a disorder in his body image.

Another patient, with a gunshot wound in the left anterior parietal and posterior frontal lobes, showed motor and sensory disturbances in the right side of his body. The sensory changes could be elicited only under special conditions and were expressed as a prolongation of the periods of sensory adaptation and after-sensation. With paired equal stimulation, sensation was apparently enhanced on the affected side. With paired simultaneous stimulation in which a strong stimulus was applied to the left side and a weak stimulus to the right side, there was reduction of sensory adaptation time, and even extinction of sensation.

These observations reveal that most of the disturbances in these patients were due to normal mechanisms (such as rivalry with resultant dominance, sensory adaptation and after-sensations) which became apparent under pathologic conditions, such as lesions in the parietal lobe. Why these mechanisms become so apparent is not clear. Without the foregoing considerations and special investigations of sensations, most of the symptoms could not be explained, because routine neurologic examination for sensation revealed an essentially normal status in both these patients.

J. PRESTON ROBB

On the Pathogenesis of Causalgia in Peripheral Nerve Injuries. Nathan, P. W.: *Brain.*, **70:** 145, 1947.

Causalgia is a pain described as burning, like fire, like a red hot wire. It may vary in intensity from the most trivial burning to a state of torture. There may be other associated phenomena. The author produces evidence to show that causalgia may occur in complete lesions of peripheral nerves. He also produces evidence to show that in peripheral nerve injuries with causalgia, the pain impulses arise in the central end of the damaged nerve, and they do not arise in the nerve endings of the region which is felt to be painful.

He concludes that the most satisfactory hypothesis of the genesis of causalgia is a stimulation of the somatic sensory axons at the site of the lesion; this stimulation is caused by efferent impulses coming down the sympathetic outflow; the site of the lesion forms an artificial synapse where this stimulation occurs; the pains due to impulses arising at this synapse are referred by the patient to the area of distribution of the sensory nerve. J. PRESTON ROBB

Toxic Effects of Intrathecal Administration of Penicillin. Walker, A. E.: *Arch. Neurol. & Psychiat.*, **58:** 39, 1947.

Two cases illustrating types of neural reaction to intrathecal administration of penicillin—radiculitis and myelopathy associated with chronic leptomeningeal thickening—are presented.

It is known that intrathecal medication may produce leptomeningeal reactions, and many substances when injected into the subarachnoid space, have been found to be responsible for such alterations in the arachnoid. That penicillin may give rise to the same result is not

unexpected. Whether the primary effect of the drug was on the leptomeninx or the nerve tissue could not be determined from this study.

In the cases presented, and in most of the similar cases presented in the literature of complications following the intrathecal administration of penicillin, the amount of penicillin administered at one time was greater than is usually advised. Whether the concentration or the total amount given is the more potent factor in producing the complications is difficult to determine, for usually a large dose is associated with a high concentration of the drug. It would seem that intrathecal administration of penicillin in doses not in excess of 20,000 units and well diluted with spinal fluid is unlikely to cause this complication. There is no evidence that the use of large amounts is more effective in the treatment of purulent meningitis than the use of moderate doses.

J. PRESTON ROBB

Dermatology

Calcium and Growth in Aging and Cancer. Lansing, A. I.: *Science*, **106**: 187, 1947.

Calcium increase with age has been demonstrated in a wide variety of organisms. There is marked increase in the calcium content of cell periphery or cortex. It has been suggested that a permeability decrease might result in the accumulation of katabolites producing in turn senescent changes. There has been some experimental support for this view, rotifers in a low calcium medium, for instance, living longer than others, while removal by sodium citrate of calcium from cells of rotifers resulted in marked increase in longevity. Experiments have resulted in general agreement that starvation increases longevity by extension of the period of growth, and it is apparent that cessation of growth may set off the ageing mechanism, or that the process of ageing is a consequence of the change which occurs with the cessation of growth. One would expect to find the opposite situation in cancer, which may be regarded as a very youthful and vigorous tissue with tremenduous and uncontrolled growth capacity. Cancer tissue has repeatedly been shown to be remarkably low in calcium. Methylcholanthrene-induced epidermal hyperplasia shows 50% reduction of total calcium below that of normal epidermis, and in squamous-cell carcinoma there is a reduction of approximately 60% from the precancerous hyperplastic epidermis. The ultrafilterable calcium fraction of squamous-cell carcinoma in mice was sharply reduced on both an absolute and relative basis. The calcium-binding complex which changes in cancer to limit calcium uptake is believed to be located in the cell cortex. It is of further significance in this connection that normal cells are more readily separated by micromanipulation when in a calcium-free medium than when in balanced salt solution, also that cancer cells are similarly more readily separated than normal cells, it being suggested that the decreased adhesiveness results from a local calcium deficiency.

D. E. H. CLEVELAND

Histoplasmosis. A Review of the Cutaneous and Adjacent Mucous Membrane Manifestations with a Report of 3 Cases. Curtis, A. C. and Grekin, J. N.: *J. Am. M. Ass.*, **134**: 1217, 1947.

Until recently histoplasmosis has been thought of as an extremely rare disease of tropical origin, but a rapidly increasing number of cases in the United States has lately been reported. At present the central portion of the country appears to be the endemic focus of the disease. Of almost 70 cases described in the past 8 years 41 were from this area. In most instances the disease has been systemic and generalized, but involvement of skin and adjacent mucous membrane is common, and apparently at times the disease may be localized. *Histoplasma capsulatum* is distinguished from other fungi in the fact that it primarily invades the reticuloendothelial system. The authors describe its morphological and cultural characteristics and advise that when it is suspected, biopsy specimens, peripheral blood, aspirated sternal marrow and abscess fluid should all be cultured. The value of the skin test with histoplasmin is not securely established but it is considered that negative reactions furnish a useful screening device. Three cases are described in detail: one showed generalized involvement including cutaneous lesions which terminated fatally; a second presented lingual and cutaneous lesions of 12 years' duration, and was clinically cured by sulfadiazine, but several months later the organism could still be demonstrated in microscopic sections; a third case presented penile lesions with regional lymphadenopathy of 3 years' duration, responded well to sulfadiazine and 2 years later the organisms could not be found in the lesions. Twenty-one other cases with cutaneous lesions had also been studied by the authors. In the differential diagnosis syphilis, epithelioma, tuberculosis cutis, blastomycosis, chromoblastomycosis, granuloma inguinale, leishmaniasis, moniliasis and sporotrichosis are among the conditions to be considered.

D. E. H. CLEVELAND

Junction Nevus—Nevocarcinoma. (The So-Called Melanoma Group.) Sachs, W., MacKee, G. M., Schwartz, O. D. and Pierson, H. S.: *J. Am. M. Ass.*, **135**: 216, 1947.

The term melanoma is not favoured, as it is loosely applied to any type of pigmented nevus and it is ambiguous as it refers to a certain type of nevus and the malignant growth which develops from it. The terms junction nevus and melanocarcinoma are more descriptive and specific. Junction nevus is so called because it arises at the junction of epidermis and cutis. It is usually a pea-sized, flat or slightly raised, non-hairy, smooth lesion, deep brown to black in colour. It may occasionally be much larger, elevated, verrucous, hairy or non-pigmented. The differential diagnosis includes the consideration of a number of other diseases, but a biopsy is of prime importance in making a conclusive diagnosis. Even though the microscopic diagnosis proved to be junction nevus the authors have never known cancer to develop as a result of biopsy, nor has biopsy increased the malignancy of existing nevocarcinoma. Since many benign lesions which may simulate junction nevus occur in sites where suspected lesions cannot be widely excised without causing serious disfigurement, the authors consider that such operative procedure is never justified without microscopic diagnosis. Many other authorities also agree that there is practically no possibility of disseminating tumour emboli by careful biopsy. The histologic diagnosis is easy and only other pigmented nevi and the transitional changes between nevus and cancer require differentiation. The intraepidermic type of epithelial nevus shows no microscopic similarity to the junction nevus, but the intradermic nevus while resembling the junction nevus is not connected with the epidermis and shows none of the characteristic cell-nests which interrupt the basal cell margin in the junction nevus. Malignant change in a junction nevus results in nevocarcinoma. The alteration in the character of the cells or extension of the process may be so gradual as to give no clinical evidence of its occurrence. Subdivision into early and late stages is based on the degree of malignancy found rather than on the time factor. Alteration in size, colour or surface, ulceration, inflammation, scaling, bleeding, crusting or tenderness may be observed. Many of these changes are seen in late nevocarcinoma, although it may be of brief duration. In early nevocarcinoma microscopically viewed some embryonic or anaplastic cells are present and mitotic figures are usually visible. In late nevocarcinoma practically all the cells are anaplastic. In the authors' opinion the change to malignancy may occur with or without trauma, which may be slow and gradual as from pressure, friction and other irritation, or rapid and specific as by a blow.

The epidermic pigmented nevus is superficial, raised, verrucous, light to deep brown, from one to several centimetres in size and usually is coarsely haired. The common mole is usually an intradermic nevus, has a pre-

dilection for the face, and is often multiple. The size ranges from a few millimetres to one or more centimetres. The colour may be that of normal skin or brown. The surface is usually smooth and may be hairy or non-hairy. It usually develops early in life and remains stationary. The blue nevus, of mesodermal origin, is impossible to distinguish from a junction nevus without biopsy, and malignant change has not been observed. As the junction nevus is a precursor of nevocarcinoma it should be excised, but this does not call for excision of more than the nevus itself. In early nevocarcinoma the prognosis while serious is not hopeless. Wide excision followed at times by radiation therapy is required.

D. E. H. CLEVELAND

Pathology

Nodular Inflammatory and Degenerative Lesions of Muscles from Four Hundred and Fifty Autopsies. Clawson, B. J., Noble, J. F. and Lufkin, N. H.: *Arch. Path.*, **43**: 579, 1947.

During a study of muscles obtained from 450 routine autopsies, nodular myositis and muscular degenerative lesions were encountered fairly frequently. Nodular myositis was present in at least one muscle in 26.2% of cases. Degenerative lesions were present in even greater numbers—atrophy 42%, sarcoplasmic changes (Zenker degeneration) 33.7% and nuclear changes in 35.1%. The diaphragm, sternocleidomastoid and intercostal muscles were most frequently found involved. The distribution was about equal between the sexes, with the occurrence in both sexes being greater in the sixth to ninth decades. An analysis of the diseases causing death revealed that apart from acute rheumatic fever and rheumatoid arthritis in which muscular lesions were present in 100% of cases, there appeared to be no particular disease or group of diseases which increased the frequency of the lesions. They were present in 29.0% of 93 cardiac cases, 31.9% of 144 non-infectious diseases, and 15.5% of 77 infectious diseases. Highest incidences were found in bacterial endocarditis (40%), cirrhosis of the liver (55.5%) and gastro-intestinal diseases (45%).

Although inflammatory and degenerative lesions were often found together in the same muscle, they were also found separately and it is suggested that they both arise due to the same cause and that the degenerative lesion followed the inflammatory.

W. B. LEACH

Nonspecific Myocarditis. Analysis of a Series of Thirty-six Cases. Marcuse, P. M.: *Arch. Path.*, **43**: 602, 1947.

The author presents a survey of 36 cases of nonspecific myocarditis encountered among 3,800 consecutive autopsies. In none was there any evidence of endocardial or pericardial involvement, but inflammatory lesions or organs other than the heart were found in 35 of the 36 cases. These included (1) lungs only—18, (2) lungs and other organs—8, (3) organs other than the lungs—9. Bronchopneumonia and interstitial pneumonitis accounted for most of the pulmonary diseases associated with the myocarditis. Clinically, objective signs of cardiac disease were present in 9 cases and in 13 there was an associated leukocytosis of between 10,000 and 20,000.

The lesions are divided into diffuse, focal and minimal types. The basic feature of all is an infiltration with leukocytes (polymorphonuclears, lymphocytes and plasma cells in varying proportions). Other parenchymatous changes are regarded as secondary to the infiltrative lesion. Although it is significant that in all but one of the cases there was an extracardiac focus of disease, no other constant findings are put forward that could be considered as possible causal factors. Evidence is against drug sensitization since in only 6 cases was there a known use of sulfonamides and in 17 cases it could be ruled out altogether. The authors are of the opinion that, in view of the presence of a focus of infection elsewhere, the cause of the myocarditis is the response of the interstitial tissues to a bacterial toxin acting from a distance. Bacterial studies of the myocardial lesions were done in three cases only and are therefore not significant.

W. B. LEACH

Industrial Medicine

Occupational Skin Diseases. Bulmer, F. M. R.: *Canad. Chem. & Process Industries*, **31**: 524, 1947.

From an industrial viewpoint, occupational skin diseases, which comprise at least 60 to 70% of all cases of occupational disease, are very important. Although seldom fatal they are responsible for much suffering and for a considerable amount of lost time. The amount of skin damage varies with the individual. The causative agents include mechanical action such as friction and pressure; physical agents such as heat, cold, electricity; biological agents such as bacteria and parasites; and chemicals. Of these the most common causative agents are chemicals and they act on the skin in many ways. The resultant skin diseases can be divided into two groups: (1) those due to contact with primary irritants; and (2) those due to contact with specific irritants. Details are given of the following materials in their rôles as causative agents of occupational skin disease: oils and greases, solvents, acids and alkalis, chromic acid and its salts, nickel, synthetic resins and waxes, cyanides, rubber ingredients, certain drugs and military explosive chemicals.

The author is of the opinion that the prevention or control of occupational dermatitis can be accomplished only through co-operation between workers and management. He stresses the importance of, selection of workers in plants where definitely irritating or sensitizing materials are used; proper cleansing at end of work period and adequate washing facilities; substitution of non-irritating or less irritating materials in the factory, protective clothing, protective creams, reduction to the minimum of the amount of skin contact with offending agents, and changing the worker's job when necessary.

Treatment of an occupational dermatitis should be undertaken by a physician; a dermatologist should be consulted when necessary. Information regarding the materials to which an affected worker has been exposed is of great assistance to the physician. This can be supplied by the chemist.

MARGARET H. WILTON

Traumatic Heart Conditions. Gilbert, N. C.: *Indust. Med.*, **16**: 223, 1947.

The danger of damage to the heart by non-penetrating wounds of the chest is discussed in this article. Cardiac contusions may result from any sudden, sharp blow upon the chest. Steering wheel accidents are a frequent cause. In this connection also, falls from a height have been reported. The heart may be seriously injured even though there is no external evidence of injury and no fracture of ribs or sternum. The author reviews the literature on this subject outlining initial cardiac symptoms following various accidents and their possible consequences; these may be slight or serious, sometimes fatal. The degree of injury depends upon several factors but especially upon the extent to which the sternum is pushed in and the heart compressed, and whether the heart is expanded in diastole or contracted in systole, at the moment of the impact.

The anatomical findings of certain fatal cases at autopsy are described. The author however, emphasizes less extensive injuries which can eventually become quite as serious. Among the results of cardiac contusion, less immediate in symptoms and findings, are sub-pericardial hæmorrhages, or hæmorrhages into the pericardial sac. The myocardium may be bruised or broken. There may be hæmorrhages into the valves or the valves may be torn. There may be sub-endocardial hæmorrhages. The author stresses the necessity of considering all future possibilities no matter how slight the symptoms seem at first or how mild the shock experienced. It is his contention that every case of possible chest injury should have an electrocardiogram at once, repeated on succeeding days.

MARGARET H. WILTON

OBITUARIES

Dr. F. H. Bradley died recently in Sherbrooke, Que.

Dr. Alexander Douglas Campbell, died at the age of 65 on October 18. He retired last April from his duties as superintendent of the Weyburn Mental Hospital, which position he had held since 1930. Dr. Campbell graduated from Toronto in 1907 and came to Saskatchewan in 1911, entering partnership with Dr. Leonard Panton of North Battleford. He practiced in obstetrics till 1916 when he joined the mental hospital staff, going to Weyburn Mental Hospital when it opened in 1921. Surviving him are two sons and a daughter.

Dr. Hugh W. Crawford, 43, a native of Medicine Hat, Alta., died recently in Wellesley, Mass. He was a graduate of the University of Alberta and the University of Toronto. Dr. Crawford was also a member of the Miramichi Anglers Association of New Brunswick.

Dr. Harold Beveridge Cushing, died October 31, in the Western Division of the Montreal General Hospital. He was 74 years old. Dr. Cushing entered the hospital the day after he completed hospital rounds on "doctors' day" at the Alexandra Hospital as was his custom for many years.

He had devoted his 50 years in medicine to the study of children's diseases and was a member of almost every medical association in North America. He was the first Montreal doctor to specialize in children's diseases.

In the early nineties, Dr. Cushing was a demonstrator in botany at McGill University. Because of lack of appointment in natural history at that time he left this field to study medicine. Dr. Cushing's interest in the study of nature never waned and one of his favourite pastimes was chatting with gardeners at the institutions he visited.

He was the son of Lemuel Cushing, a Montreal lawyer and former M.P. for Argenteuil County, and the former Ellen M. MacDougall. Dr. Cushing was born in Montreal and obtained his B.A. degree from McGill in 1892 and his degree in medicine six years later.

After his graduation, Dr. Cushing interned at the Royal Victoria Hospital and became a demonstrator in medicine at McGill on completion of his internship. Realizing the need of specializing in children's illnesses, Dr. Cushing became associated with the Children's Memorial Hospital in 1903. For 30 years he campaigned to have McGill University open a department for pædiatrics. He became the first head of this department in 1938 and because of the ruling calling for retirement at 65, he retired in 1939.

Dr. Cushings's appointments included: Emeritus professor of pædiatrics at McGill University; honorary physician-in-chief of the Alexandra Hospital; consultant for the Crippled Children's Hospital and physician to Shriners' and the Royal Victoria Hospital. He was a member of the Canadian Medical Association, the Royal Society of Physicians and Surgeons in Canada; and the Canadian Society for the Study of the Diseases of Children.

He was also a member of the Historical Society of Argenteuil County, the Montreal Medico-Chirurgical Society, the Royal Montreal Curling Club and the Westmount Bowling League and Nu Sigma Nu, Alpha Omega Alpha and Sigma XI.

When poliomyelitis swept Canada last year, Dr. Cushing came out of retirement to guide the medical field in handling the epidemic.

As emeritus professor of pædiatrics at McGill he gave one lecture a year to final year students. At the height of the polio epidemic while making his rounds at the Alexandra Hospital Dr. Cushing was in the habit of gathering young doctors around him to instruct them in nature study, describing the habits of the birds in the hospital's grounds. Then he would lead junior doctors through the wards offering advice, comments and criticism.

When asked to comment on the opening of his 50th year in medicine some time ago Dr. Cushing remarked: "I am a happy man and contented. I love children and I like to reminisce".

Dr. Cushing is survived by a daughter, Mrs. Richard Harbert, wife of Dr. R. G. M. Harbert; a brother, James Cushing, and a sister, Miss Florence Cushing.

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An Appreciation

There are probably few medical men in Canada, and certainly very few in Montreal who did not come in contact with Dr. Cushing at some time. And yet he was a specialist and might hardly be expected to have so wide an influence. There were two main reasons for this. Whilst he was known as a pædiatrician he also devoted much time to the so-called infectious diseases and so entered the field of general medicine. This was particularly evident as regards poliomyelitis, in the study of which he became outstanding. But his chief strength lay in his breadth of mind and his capacity to survey a subject in all its aspects. And yet he was never diffuse. He possessed rather a capacity for setting forth the essentials of a subject with a clarity and force which might at first seem magical, but which was actually due to his command of simple language. He realized how few words are really necessary to state facts, and more, he used only few. Obviously he could not carry in his mind all the detail of all medical developments, but his gift of discrimination again allowed him to select the essential and to put things in their proper perspective. He wrote very little, however. Whilst he was in great demand for addresses these were almost always from very brief notes. They were delivered so faultlessly that a verbatim report would have needed very little alteration, for he could trust to his memory without a manuscript.

To go round the wards of the Alexandra Hospital (for infectious diseases) with him, was to be shown the natural history of disease in the clearest and most impressive manner. Out of what seemed to be only "run of the mill" cases he would always extract the unusual or at any rate the striking. In a medical meeting he never seemed to be particularly intent on the proceedings, but when he got up for discussion the whole fabric of the subject was seen to be in his hands. As someone once remarked of him at a meeting: "he is always either asleep or making the best address of the evening".

His name will probably always be associated with his remarkable ability in diagnosis, as well as that most difficult art, prognosis. He seemed to size up a case at a glance, but said nothing until he had the significant facts before him, and then frequently he would unhesitatingly assign to the fever, or rash, or illness its period and outcome, and he was very seldom wrong. Possibly some of the stories of this power of his are apocryphal, but not many. As a consultant therefore he was in constant demand, and if his rather detached manner was sometimes a little discouraging it was as unintentional as his conclusions were unshakeable. He did not attain his full powers at once. Some who were students under him in his younger days were not impressed by his teaching. Actually I think he had a certain amount of diffidence to overcome. But there was never any question of his mental power and as soon as he found himself he rose steadily to the high position which he held in the last 25 years of his life.

It has been said of him that he was the greatest gift to Canadian medicine since Osler: the generalization has its merit so far as one can make comparisons of this kind. As a clinical teacher he was not excelled, but all his penetrating clinical judgment would have been worth little without his firmness of character. No one obtruded himself less, but no one was less easily persuaded against his judgment. He had the gift of reticence, but he commanded the respect and affection of a large circle of friends both in his profession and

in society generally. With his death we have lost a man of rare powers, who knew how to use them only for good. H.E.M.

Dr. Emile Frederic Fluhmann, a former resident of Montreal, died on September 21 at his home in Ottawa, in his 81st year.

He was educated at the old Laval University in Montreal. Later he practised medicine at Roberval, Que., after which he was appointed medical officer for the Price Brothers Paper Company at Kenogami, Que. He also acted as C.N.R. medical officer at both places.

He retired in 1929 due to ill health and lived in Montreal until 1946, when he moved to Ottawa. He was a member of the Masonic Order at Quebec City.

He is survived by his widow, a son, a daughter, and four sisters.

Dr. Thomas Andrew Fraser, aged 34, died at Sunnybrook Hospital, Toronto, on October 10. Stricken with nephritis while on active duty in Holland, he was invalided home in June, 1945. He held the rank of captain.

Born in Clinton he graduated in arts from the University of Toronto in 1936. In the following year he took his master's degree. He received his M.D. in 1940. Winner of the Ellen Meikle Fellowship, he did postgraduate studies at Yale.

Surviving are two sisters.

Dr. George William Henry French, aged 84, died suddenly on October 11 in Toronto.

He was educated in England and was a former house surgeon and physician at St. Mary's Hospital, London. He was senior surgeon at Horssey Central Hospital, London, for 20 years. Retiring nine years ago, he came to Toronto and was a member of the Academy of Medicine. Besides his daughter, he leaves a brother.

APPRECIATION OF THE LATE DR. H. W. HILL

[*The following tribute to Dr. H. W. Hill has been kindly sent us by Dr. H. H. Murphy of Victoria, B.C. It appeared in the "Health Bulletin" of the B.C. Public Health Service.*—EDITOR.]

A man who taught public health to many men and women, who in turn carried his philosophy to far-flung communities in North America, passed away on May 23, 1947, in his 75th year. This great pioneer was Dr. Hibbert Winslow Hill, who as director of the Vancouver General Hospital Laboratory from 1925 carried out public health work, and later assumed directorship of the Provincial Board of Health Laboratory when it was officially formed in 1931. He remained in this position until ill-health forced him to retire in 1935. In this ten-year period he was also Professor of Bacteriology at the University of British Columbia and Health Officer for that area. At the time of his death Dr. Hill was living in Hackensack, Minnesota.

Dr. Hill, who was born in Saint John, N.B., September 16, 1871, has an impressive list of "firsts" on his record. In 1893 at the age of 22, he was recognized by many as the first professional bacteriologist in Canada, was the first man designated as official epidemiologist in North America when he was appointed to the Minnesota State Board of Health in that capacity in 1908, and in 1913 became the first dean of the Faculty of Public Health at the University of Western Ontario. He has also been credited with originating the term "epidemiology", and was one of the charter members of the "Bug Club", forerunner of professional bacteriological associations in America today.

Besides lecturing at the University of British Columbia, he was at various times a staff member, lecturing in bacteriology and pathology, at Toronto University, the University of Western Ontario, Harvard University, and the University of Minnesota. Through his teachings at these universities and work on many health boards in the United States and Canada, this pioneer proved himself an enthusiastic, capable, and successful missionary for public health.

He was one of the earliest advocates of the "puncture" method of smallpox vaccination, and devised new methods for counting and watching the growth of colonies of bacteria. "Who's Who in Canada" also credits him with development of porous tops for "petri" dishes and methods for filtering sera under pressure. He carried out intensive research in diphtheria, typhoid fever, tuberculosis, and nutrition.

One of Dr. Hill's beliefs was that public health must be "popularized" through the medium of speeches and newspaper and magazine articles to be fully understood by the public. Consequently during his life he delivered close to a thousand addresses on public health subjects, and wrote several hundred "popular" articles, in addition to making frequent contributions to scientific and medical journals. He wrote several books, "Sanitation for Public Health Nurses", "America and the New Era", "Clean Milk", and "The New Hygiene", and collaborated on "Microbiology".

Dr. Hill once described public health this way: "It is the latest and greatest of the sciences and arts, because it includes all the others to date. It exists as a twentieth century vision, already partly realized, of a whole world in which each individual fits his own smaller world and his own smaller world fits him". In more technical phrase, public health seeks the adjustment of every man to his surroundings, and his surroundings to every man.

Dr. Arthur Wellesley Hotham, aged 81, died at Strasbourg, Sask., September 21.

He taught school at Boggy Creek school district during the 90's to assist him to attend medical school. He graduated from Trinity Medical College, Toronto in 1898.

Dr. Hotham came to Strasbourg in 1905 and practised here for 21 years. Then he accepted a position with the North Battleford mental hospital. In 1931 he went to Earl Grey and during the war practised at Montmartre and Semans. Upon his retirement last year he moved back to Strasbourg.

He is survived by two daughters, two brothers and three sisters.

Dr. Walter Bingham Kendall died on October 17 at his home in Gravenhurst, Ont., following a heart seizure. He was 76 years of age, and has been retired since February, 1938, when he suffered a stroke.

Born in Bradford, Dr. Kendall received his education in Toronto. Graduating in 1897 from the Ontario College of Pharmacy, he joined the staff as lecturer, continuing in this position for eight years. Entering Trinity Medical School, he was a graduate in the class of 1906, and later took postgraduate study in Edinburgh, where he received the degree of F.R.C.S. Shortly after his return to Canada he was appointed chief at the Muskoka Hospital for Consumptives.

Organizer of Gravenhurst Rotary Club, he was the first president. He was an active worker and elder of the United Church.

Surviving are his widow, a son, a daughter and a brother.

Le Dr J. Emilien Marcil, attaché à l'Unité Sanitaire de Drummondville, Que. depuis plusieurs années, est décédé le 13 septembre à l'hôpital Ste-Croix à l'âge de 48 ans, après une longue maladie.

Il laisse dans le deuil son épouse, quatre filles et un fils.

Dr. Mark W. Pascoe, of San Gabriel, Calif., died on August 23 following a brief illness. Born in Solina, Ont. in 1871, he attended Bowmanville High School, McGill University and Trinity College, Toronto. In 1900 he went to Ottumwa, Iowa, where he practised surgery and general medicine for one year. He then lived in

Los Angeles for a short time before going to Taft in Kern County where he built a hospital. He practised here until his retirement in 1928 when he took up research work in 1936 at San Gabriel.

He is survived by his wife, one brother and three sisters.

Dr. Cecil Rowland, aged 49, died suddenly on October 7 in Vancouver. He was born in Coquitlam and educated at McGill and Toronto Universities. He practised at Kimberley for 12 years before coming to Vancouver.

Surviving him are his widow, two sons, his father and one brother.

Dr. Frank W. Skaife died on October 2 at his summer home at Lake Memphramagog, Que. He was in his 86th year.

Born in Montreal in 1861, he was educated at St. Mary's College and the Montreal High School and graduated in medicine from McGill University. He also studied for a period at the Ontario Agricultural College at Guelph, Ont., and in England.

Dr. Skaife practised medicine for many years in California, living in San Francisco. After retiring, he took up residence in London, England.

Le Dr Alexandre Thibodeau est décédé récemment à San-Francisco où il pratiquait sa profession.

Il était né en 1877 à S.-Jacques l'Achigan, Québec. Il avait fait ses études classiques au collège de l'Assomption et ses études médicales à l'université Laval de Montréal, où il avait gradué en 1903. Après un séjour de deux ans en Alaska, il s'était établi à San-Francisco où sa carrière professionnelle fut couronnée de succès et où il jouissait de la considération de tous. Il était membre de la colonie française et de la société américaine.

Durant vingt ans il avait fait partie du corps médical de l'hôpital Français, dont il a été le président. Il était aussi l'un des médecins du consulat général de France à San-Francisco et il avait servi comme officier médical dans l'armée américaine durant la première grande guerre.

En 1926, le gouvernement français avait reconnu ses services en le nommant chevalier de la Légion d'honneur.

Dr. Harold M. Tovell, aged 60, died on October 16 at Toronto General Hospital after a brief illness. Dr. Tovell's education, first at St. Andrew's College and later at the University of Toronto, prepared him for his entrance to Columbia University, from which he graduated in medicine in 1912, later doing postgraduate work in New York and Germany.

A pioneer radiologist in Canada, Dr. Tovell was for many years radiologist to both the Wellesley and St. Michael's hospitals and assisted in organization of the x-ray department at the Hospital for Sick Children. Prior to this he was on the teaching staff in clinical medicine at St. Michael's and Toronto General Hospitals.

Pursuing wide interests in the arts and in the work of young Canadian artists, Dr. Tovell was keenly interested in the development of the Group of Seven and was closely associated with the Art Gallery of Toronto. In addition, he was actively engaged in the work of the National Council of Education and the Royal Ontario Museum of Archæology.

He is survived by his widow, and four sons.

Dr. Robert McPherson Turner, aged 66, died on October 6 at the Toronto General Hospital.

Since his graduation from the University of Toronto in 1905, Dr. Turner had practised medicine in Toronto. He was associated with the Toronto East General Hospital, and was a member of the St. George's Golf Club.

He was twice married, his first wife dying in 1939. Surviving are his widow, a son, Dr. D. L. Turner, by the first marriage; two sisters and two brothers.

Le docteur Edouard Verdon est décédé le 15 octobre à Montréal à l'âge de 67 ans.

Né à Ville S.-Laurent, il avait fait ses études classiques au collège de Saint-Laurent, puis avait suivi les cours de la faculté de médecine de l'université Laval de Montréal. Reçu médecin en 1902, il avait pratiqué tout d'abord à Napierville, pour être ensuite attaché, durant une quinzaine d'années, à l'Institut Bruchési de Montréal.

Il laisse sa femme, deux fils et deux filles.

NEWS ITEMS

Alberta

The Defence Medical Association, Alberta Division, held its annual meeting in Edmonton on October 17, 1947. Resolutions were drafted for presentation at the annual meeting of the Dominion body to be held in Ottawa, November 7 and 8. Dr. D. R. Easton as Representive, and Dr. W. C. MacKenzie as a member of the Executive, and Major W. MacLaren as Secretary were elected to attend the meeting.

A recent book publication from Alberta is that fine work of Dr. Heber C. Jamieson of Edmonton, entitled "Early Medicine in Alberta". This valuable history of the first seventy-five years of medicine in this Province will become even more valuable as time goes on. The book is well illustrated and detailed accounts are found throughout its some two hundred pages. Dr. Jamieson has given much to the annals of medical history and this most recent work is an accumulation of a great deal of work. The book may be obtained through the Registrar's office in Edmonton.

Following the summer vacation, the many medical societies and medical reporting clubs, the University faculty and departmental clubs are holding regular meetings, all of which are a great asset to the advancement of medical progress, and an inspiration to the younger men of the profession throughout Alberta.

The Edmonton Academy of Medicine, had as its guest speaker, Dr. R. F. Bannock, Professor of Clinical Medicine at the University of Washington, Seattle. An excellent illustrated address was given on "The Differential Diagnosis and Treatment of Jaundice". Dr. Bannock brought this subject into a very understandable light and the Executive members of the Academy had the wisdom to have the address printed for its members, a copy of which may be obtained from the Registrar's office.

W. C. WHITESIDE

British Columbia

The extensions to the buildings of the B.C. Cancer Institute, situated in Vancouver, are going ahead apace, and this very active and busy institution will shortly have considerably increased facilities for its work, which has grown very greatly in the past few years.

Extensions are also in progress, to the buildings of the Provincial Clinic for Diseases of the Chest, on 10th Avenue and Willow Sts. in Vancouver. The great advances that have been made of late years, in chest surgery, have led to a very great increase in lung and heart surgery in British Columbia, and the new buildings will provide adequate facilities for this.

The B.C. Hospitals Association, at its recent annual meeting, passed a resolution, urging on the Government of B.C. the advisability of instituting a scheme of free hospitalization for all the citizens of the Province, this

to be paid for by a tax for that purpose. It is to be hoped that a coincident scheme of hospital construction will be developed at the same time, or even in advance of such a step. Both would be extremely desirable, if conceived on a sound financial basis.

The recent visit to Vancouver of Dr. A. H. Gordon, the eminent Montreal internist, was more than welcome. Acting on the definition of gratitude so well-known to us all as a lively sense of favours to come, the Vancouver profession proceeded to fill every available minute of Dr. Gordon's time, with clinics, luncheons, addresses, and the like, to the saturation point. Dr. Gordon, the most generous of men, seems to have adopted the slogan of one of our national gasoline agencies—"service with a smile", and gave freely from his well-crammed store of knowledge, adding, if that were possible, to his reputation as a teacher. His reminiscences were, as in any address given by this most able speaker, entertaining and delightful to a degree.

The Red Cross maintains a very active campaign for blood donors. With the tremendous and ever-growing demand for blood from the hospitals, all over B.C., the problem of securing a steady supply is no small one. The student body of the University of British Columbia is supporting the movement actively, and with the President of the University, Dr. N. A. McKenzie, setting the example as the first donor, it is expected that of the 8,000 or 9,000 students now enrolled at the U.B.C., a large majority, if not all, will contribute. The object aimed at, is two pints per student. This would do a great deal to make the minds of those in charge of the work easier.

The police of Vancouver have been complaining of the unwillingness of medical men to take emergency calls at night, especially in the poorer quarters, and state that they have records of many cases where such refusal on the part of the medical men has led to hardship. Dr. A. J. MacLachlan, Registrar of the College of Physicians and Surgeons, has the matter under investigation. Unless the city is willing to provide for emergency medical service at all hours of day and night, to take care of police calls, accidents, and the like, it is not easy to see how the profession as a whole can either be dragooned into doing this work, which is mostly quite unremunerative, and often productive of much grief, or blamed for avoiding it. At any rate, both the B.C. College of Physicians and Surgeons and the Vancouver Medical Association are looking into the matter. J. H. MACDERMOT

Manitoba

The degree of Fellow of College of American Pathologists has been conferred on Dr. T. H. Williams, director of Laboratories, Deer Lodge Hospital. He received the honour at a convention of pathologists in Chicago, October 27. Dr. Williams spent many years as a medical missionary in Szechwan, West China, before returning to Manitoba.

Manitoba will be represented at the annual meeting of the Royal College of Physicians and Surgeons of Canada in Ottawa on November 28 and 29 by Dr. J. M. Kilgour who will speak on "Chronic Hepatitis" and Dr. C. W. Burns who will preside at a meeting of the Division of Surgery. ROSS MITCHELL

New Brunswick

Dr. A. W. Blair of Regina was in New Brunswick lately beginning a coast to coast survey of present Canadian activities in the field of cancer research, treatment and education. When the survey is completed the results will serve as a basis for a program of the Cancer Institute of Canada, of which Dr. G. E. Richards is President. During Dr. Blair's visit in New Brunswick he was assisted by Health Minister, Hon. Dr. F. A. McGrand, Chief Medical Officer Dr. J. A. Melanson and Dr. D. F. W. Porter, chief of Provincial Hospital services and Miss Marion Belding of the N.B. Branch of the Canadian Cancer Society.

Dr. G. E. Maddison, lately of the tuberculosis service of the D.V.A. at Lancaster Hospital has been appointed Director of Tuberculosis Control under the Provincial Department of Health.

Dr. John L. Thompson who has practised at Grand Manan, N.B. since his discharge from the armed forces has now begun practice in Saint John.

Dr. Earl Grant was the special speaker at the October meeting of the Saint John Medical Society. His subject "Plastic Surgery". The importance of plastic surgery is increasingly admitted in view of the mutilation so common in highway accidents and subsequent to Industrial traumas. Another field is that following destructive malignant lesions where loss of tissue remains after arrest of the malignancy by radiation or surgery.

Dr. J. R. Nugent, chairman of the N.B. Medical advisory committee on cancer attended the recent week long sessions on cancer treatment and cancer research at Toronto.

Dr. D. H. MacKenzie has established his practice at Grand Manan, N.B.

At the October meeting of the Pictou, N.S. County Medical Society, the invited speakers were Dr. J. K. Sullivan, whose subject was "Prostatitis" and Dr. G. W. White, who spoke on "Obstetrical Blood Loss". Both speakers are members of the attending staff of the Saint John General Hospital.

A number of New Brunswick physicians have completed short refresher postgraduate studies in the United States. Dr. W. O. McDonald, Dr. A. L. Donovan and Dr. W. Murphy attended courses in Boston and Dr. F. D. Wanamaker studied pelvic surgery at Chicago.

A. S. KIRKLAND

Nova Scotia

The Dalhousie University Refresher Course held during October was one of the most successful in the twenty-five years since their inauguration. Lectures by distinguished physicians from Canada and the United States, along with dry and operative clinics at the local hospitals in Halifax gave a well rounded program of five days' duration. A total of 255 physicians registered. A pleasing feature of the course was a tour through the new Victoria General Hospital.

During the course the Medical Society of Nova Scotia held its Annual Meeting, with over 200 physicians in attendance. Under its auspices a very enjoyable banquet was held at the Nova Scotian Hotel. The officers of the Association for the ensuing year are as follows: *President*—Dr. Eric MacDonald, Glace Bay; *1st Vice-president*—Dr. H. A. Fraser, Bridgewater; *2nd Vice-president*—Dr. J. J. Carroll, Antigonish; *Secretary*—Dr. H. G. Grant, Halifax; *Treasurer*—Dr. R. O. Jones, Halifax.

Particular mention should be made of the numerous industrial displays featured at the meeting.

Dr. P. S. Campbell, Deputy Minister of Health is enjoying a well earned vacation. Dr. J. Sinclair Robertson, until recently Divisional Health Officer with headquarters in Yarmouth, has been promoted to the position of Assistant Deputy Minister. Dr. J. J. MacRitchie is now Inspector of Humane Institutions.

On a recent visit to Newfoundland your correspondent found that the Director of Medical Services, Dr. Leonard Miller; the Superintendent of the Newfoundland Sanitarium, Dr. R. E. Bennett; and the Superintendent of the Hospital for Mental Diseases, Dr. Charles Roberts, were all graduates of the Dalhousie Medical School.

H. L. SCAMMELL

Ontario

Dr. Melville Watson's report of the progress made by the Board of Governors of the Physicians Services Incorporated interested the members of No. 11 District of the Ontario Medical Association in Toronto in October. Employed groups will be insured. There are two types of coverage; (1) surgical and obstetrical and (2) complete care, including medical. Fees are those of the Ontario schedule of 1946. Patients will have free choice of doctor. There is no limitation of the number of patients on any doctor's list. The plan is now in operation; offices are at 600 University Avenue, Toronto.

The evening meeting held in Sunnybrook Hospital was addressed by Dr. J. E. Carson, Brantford, President-elect of O.M.A. Like Dean J. A. MacFarlane he deplores the fact that such a small proportion of present medical students have been brought up in the country. He proposed a scheme where a final year student could be attached to a general practitioner as an apprentice in order to gain practical experience. Dr. T. C. Routley gave his "Impressions of Post-War Europe". He has visited Europe four times since the war stopped. His talk had a vivid emotional appeal. He then described the organization activities of the World Health Organization. He and a Russian delegate worked together on the rules committee. The first meeting is to be held in North America.

The auditorium at Sunnybrook is a very pleasant spot for a medical meeting. Its spaciousness is emphasized by light colours in floor, walls, furniture and hangings. The chairs are light weight and comfortable, the acoustics are excellent, the ventilation is air conditioned and there is no parking problem in the acres of driveway.

Professor Ernst Rothlin of Basle, Switzerland, addressed University of Toronto Physiological Society in October on "The Fate of the Ergot Alkaloids in the Organism". Recent investigations have further elucidated the chemical natures of the component alkaloids found in extracts of ergot; the varied pharmacological effects of the pure alkaloids have been studied. Certain derivatives of the naturally occurring alkaloids have been prepared by hydrogenation; these also have interesting pharmological characteristics. Much information has been gained by these researches. Through work of this nature the alkaloids of argot will be therapeutically employed with more intimate knowledge.

LILLIAN A. CHASE

Quebec

Un don de $5,000 vient d'être fait à l'Institut de microbiologie de l'Université de Montréal par la Légion Canadienne pour des recherches sur la poliomyélite et les maladies à virus.

L'Ecole d'Hygiène de l'U. de Montréal attend de France les Drs Henri Simonnet et A. R. Prévôt. Le Dr Simonnet donnera des cours sur la nutrition et le Dr Prévôt sur les microbes anaérobies dans leurs relations avec les problêmes d'hygiène publique et d'immunité.

Trois montréalais ont été élus "nominees" de l'American College of Hospital Administrators; ce sont, la Rév. Sr. Noémie-de-Montfort et les Drs J. E. DeBelle et J. R. Boutin.

Le Dr Roméo Boucher a été invité par la Faculté de médecine de Paris à faire quelques cours sur quelques maladies qui revêtent chez nous, sous notre climat, des particularités peu connues en Europe.

Le Dr Jean Saucier vient d'être nommé chef du service de neurologie de l'Hôtel-Dieu. Il succède au Dr A. Barbeau, décédé à Paris en juin dernier.

JEAN SAUCIER

Saskatchewan

At the last convocation meeting of the American College of Surgeons held at the Waldorf-Astoria Hotel in New York, Dr. J. B. Ritchie, of Regina, was elected a Fellow of the American College of Surgeons.

The Saskatchewan Hospital Services plan tax rate for 1948 will remain the same as in 1947. The annual fee will be $5.00 per person, with a maximum of $30.00 for a family regardless of size.

Dr. Mathew Dantow has been appointed Medical Health Officer in charge of Health Region No. 13, with headquarters in North Battleford.

Dr. Clair Rumball, formerly chief anæsthetist at Deer Lodge Hospital, Winnipeg, has recently joined the firm of Leech, McCutcheon and Bowering. While overseas Dr. Rumball was chief anæsthetist at No. 5 Canadian General Hospital, later becoming consultant in anæsthesia to the Canadian Army overseas.

Excavation work on the $7,000,000 university hospital, Saskatoon, is under way. The wing joining the medical college will be the first section of the hospital to be constructed. The hospital building when complete will be a six-storey structure containing 575 beds. The new hospital unit will be built of native stone in conformity with the general university architectural style.

B. BRACHMAN
E. W. SPENCER

General

Dr. R. J. Brown, has been appointed as medical officer for the Atlantic Region, Canadian National Railways. Formerly medical officer of treatment at Montreal. Dr. Brown will have his headquarters at Moncton, N.B., and his territory will include all lines between Riviere du Loup and Monk, Que., between Sydney, N.S., Halifax and Saint John, N.B. and all lines on Prince Edward Island.

Medical laboratory equipment originally valued at some $6,000 has been given to the Laboratory Services of the Ontario Department of Health. The transfer has been made in recognition of the generous service provided the Royal Canadian Army Medical Corps by the Ontario Health Department during the war. At a time when medical laboratory technicians were badly needed, the Ontario Government established a school and supplied the necessary instructors without charge to train Army medical personnel. At the same time the Ontario Department carried on diagnostic laboratory service for the Department of National Defence. As a result of the generous wartime assistance of the Ontario Health Department, it is now in possession of military medical equipment, with the compliments of National Defence, ranging from microscope glass slides to centrifuges and sterilizing ovens.

National Gastroenterological Association 1948 Award Contest.—The National Gastroenterological Association announces its Annual Cash Prize Award Contest for 1948. One hundred dollars and a Certificate of Merit will be given for the best unpublished contribution on Gastroenterology or allied subjects. Certificates will also be awarded those physicians whose contributions are deemed worthy. Contestants residing in the United States must be members of the American Medical Association. Those residing in foreign countries must be members of a similar organization in their own country.

All entries for the 1948 prize should be limited to 5,000 words, be typewritten in English, prepared in manuscript form, submitted in five copies, accompanied by an entry letter, and must be received not later than April 1, 1948. Entries should be addressed to the National Gastroenterological Association, 1819 Broadway, New York 23, N.Y.

Research Grants and Fellowships by the Life Insurance Medical Research Fund.—Applications for grants in aid of research on cardiovascular problems to begin in 1948 will be received by the Life Insurance Medical Research Fund up to January 15, 1948. Support is available for physiological, biochemical, and pathological research which bears on cardiovascular problems, as well as for clinical investigation in this field. It is expected that about $500,000 will be awarded for these grants. Applications for postgraduate fellowships for training in research for 1948-49 will be received by this Fund up to January 1, 1948. Preference is given to candidates who wish to work in the broad field of cardiovascular function or disease and to candidates who wish to work in institutions other than those in which they have obtained most of their experience. A doctor's degree (M.D. or Ph.D.), or the equivalent, is required. The annual stipend varies, but as a rule is between $2,500 and $3,500. Approximately 12 fellowships will be available. Later in the year, the Fund will also offer a number of student (pre-doctoral) research fellowships for 1948-49. Both grants and fellowships will become available on July 1, 1948. Further information and application blanks may be secured from the Scientific Director, Life Insurance Medical Research Fund, 2 East 103rd Street, New York 29, New York.

Roche-Organon Awards in Endocrinology.—Three awards of $500.00 each have been sponsored by Roche-Organon Inc., hormone manufacturers, for scientists in recognition of recent, outstanding research in these fields of endocrinology: animal experimentations, hormone chemistry, and clinical endocrinology. The winners of the awards are: Dr. Fuller Albright (clinical endocrinology), Dr. Robert D. H. Heard of McGill University, Montreal (hormone chemistry), and Dr. Dwight J. Ingle (animal experimentation).

The awards were presented at the 1947 Laurentian Hormone Conference held recently at Ste. Adele, Quebec, Canada.

It has been announced by Order-in-Council that barbituric acid, its compounds and derivatives are to be included in the list of drugs to be sold only on prescription by a duly qualified physician, dentist, or veterinary surgeon.

Colonel W. L. Coke, O.B.E., R.C.A.M.C., has been appointed Director General of Medical Services, Canadian Army, with promotion to rank of brigadier.

W.H.O. Helps to Fight Cholera.—The cholera outbreak in Egypt, which was first reported on September 22, 1947 provided a graphic demonstration of how the World Health Organization can co-operate with the various national health ministries and commercial drug manufacturers to battle a common menace of mankind.

Seven tons of vitally needed anti-cholera vaccine and other medical supplies were flown from New York City, on Saturday October 25, including 1,120,000 cubic centimetres of anti-cholera vaccine (enough to inoculate 750,000 persons); 3,000 syringes loaned by the City of New York; 50,000 hypodermic needles; 1,200 pounds of chemically pure salt; 1,200 pounds of glucose for intravenous injections.

The shipment from New York was part of an integrated program worked out by the W.H.O. Secretariat under the direction of Dr. Chisholm and Dr. Frank A. Calderone, Director of the Headquarters Office, New York City. Another 1,000,000 c.c. shipment was scheduled for November 1, to Egypt and Syria.

In co-operation with W.H.O., the Government of China has made available 2,000,000 c.c. of anti-cholera vaccine which is being flown by United States Army planes to Egypt. In addition, another 1,000,000 c.c. of vaccine was sent to Egypt by the following ten governments when they were advised of the outbreak: Brazil, France, Iran, Iraq, Italy, Switzerland, Tunisia, United Kingdom, U.S.S.R., and the United States.

W.H.O. Fellowships for 1947 Nearly all Awarded.—The Headquarters Office of the World Health Organization reported, as of October 31, that 192 fellowships for the current year already have been awarded. Virtually all branches of medicine and public health are represented among the doctors and other health specialists selected to study at world centres under a $1,500,000 grant from the funds of the United Nations Relief and Rehabilitation Administration for field services. An additional 200 fellows will continue this work under another grant for 1948. Among the 1947 fellows, Poland is sending 54, China 42, and Yugoslavia 35.

BOOK REVIEWS

Advances in Internal Medicine. Vol. 2. Edited by W. Dock, Long Island College of Medicine, Brooklyn, and I. Snapper, The Mount Sinai Hospital, New York. 642 pp., illust. $9.50. Interscience Publishers, Inc., New York, 1947.

This volume is an attempt to bring up to date the advances in Internal Medicine made since before World War II. The first chapters deal with the cardiovascular system. These include a full discussion of the merits of venous catheterization, angiography, and the surgical treatment of hypertension. Other chapters are devoted to the use of penicillin in subacute bacterial endocarditis and other infections. One of the more interesting chapters takes up the question of the medical aspects of aviators and deep sea diving. The final chapter has to do with nutrition and nutritional diseases in the Orient. The volume is well documented. There are few illustrations but the text is clear and in concise form. It goes a long way towards bridging the gap created by the war years.

Diseases Transmitted from Animals to Man. T. G. Hull, Director, the Scientific Exhibit, American Medical Association. 571 pp., illust., 3rd ed. $13.00. Charles C. Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1947.

The list of the contributors to this volume is an assurance of a thorough treatment of the subject. The book is divided into five sections, the largest of which deals with the diseases of domestic animals and birds in relation to man. The second section deals with those diseases of rodents and wild animals occurring in man; the third section, diseases of man which are spread by animals; the fourth section contains an interesting discussion of the animals which act as passive carriers of disease organisms; and the fifth and last section is a summary of the part played by animals in disease of mankind. This discusses the subject in tabular form and according to its importance at the present day.

This book fills a long-felt want in bridging the "no man's land" that has existed for many years between veterinary medicine and human medicine. It is a book that will be of great value to all practising doctors and should also be very useful to the sanitary inspectors and the veterinary profession.

Cancer, Diagnosis, Treatment, and Prognosis. L. V. Ackerman, Pathologist to the Ellis Fischel State Cancer Hospital; and J. A. del Regato, Radiotherapist to the Ellis Fischel State Cancer Hospital. 1,115 pp., illust. $22.00. The C. V. Mosby Co., St. Louis; McAinsh & Co., Ltd., Toronto, 1947.

The authors disclaim any suggestion that this work is encyclopædic. It is designed as a guide to further reading and study and over 1,800 authors are quoted in the references. There is not a line of "padding" in the 1,100 pages. The personal opinions of the authors are given unobtrusively and always appear as being based on sound knowledge and wide experience. Cancer is considered as a medico-socio-economic problem and the best solution offered is an increased number of special institutions in which expert surgeons, pathologists and radiologists work in co-operation.

The pathologist should be in possession of all data connected with the case and expert in such special techniques as aspiration biopsies. The surgeon "must be a pathologist, amateur physicist, physiologist and statistician as well as surgeon and philosopher". The radiologist has a definite field and his colleagues must understand that radio curability applies to many conditions but is not an alternative to surgery. "Renal and cardiac insufficiency are the only bars to operation if such be indicated". The work of the radiologist should be freed from the evil suggestion of hopelessness. The appellation "radioresistant" is often mythical as, *e.g.*, in adenoides cystica type of basal cell carcinoma.

The second part of the volume deals with regional neoplasms. Curietherapy is evidently to be considered as an adjunct to surgery in cancer of the cervix and uterus. In cancer of the tongue we have "one of the few remaining if not the last for which this form of treatment is indicated." This statement is the more significant as one of the authors is a noted radiologist. Æsthetic considerations in planning operations are put in a very subordinate place and limiting operations in order to preserve sphincter control are not approved.

Diseases of the Chest. E. H. Rubin, Attending Physician, Division of Pulmonary Diseases, Montefiore Hospital and Country Sanatorium, New York. 685 pp., illust. $13.50. W. B. Saunders Co., Philadelphia and London; McAinsh & Co. Ltd., Toronto, 1947.

Much of the older coded knowledge in respect to the diagnosis and treatment of diseases of the chest has become outmoded, and the many recent advances in this field demand incorporation in book form. This has been done in the present volume with clarity, with a point of view that reflects modern methods of practice, and with admirable balance in keeping the essentials to the fore and not blurring the discussion with theories and details of investigative work. The book is built around the fact that chest disease is no longer the province of the physician alone, but surgeon, radiologist and endoscopist now share in the field. Particularly does it reflect clinical practice in placing the x-ray in the centre of chest work. The presentation of the many categories of chest disease turns on this point, which was never achieved in the older textbooks, and this volume presents an abundance of roentgenological illustrations which gives a vividness and unity to the discussion of each condition.

The opening section deals with anatomical and radiological considerations. There follows an excellent chapter on the physiology of respiration. The discussion of symptoms and signs exhibits the original approach of this whole work—the attempt to cut away the dead wood in matters of physical diagnosis and clinical appraisal. The pneumonias are presented from the etiological standpoint and in the light of the knowledge which chemotherapy has given us. Nearly a quarter of the book deals with tuberculosis. Nowhere is the newer emphasis shown better than in the discussion of bronchial obstruction, pulmonary neoplasms in finely conceived sections dealing with heart-lung disease and chest emergencies, and in the inclusion of a section on surgical treatment.

The real value of a book of this kind lies in how adaptable it can be to clinical use, and here this work must rank high. It will serve all classes of medical people. It should bid fair to become the standard book in its field.

Dynamic Aspects of Biochemistry. Ernest Baldwin, University Lecturer in Biochemistry, formerly Fellow of St. John's College, Cambridge. 457 pp., illust. $4.75. Cambridge University Press; The Macmillan Co. of Canada, Toronto, 1947.

This attractive volume covers much the same ground as an advanced course which has been given at McGill University for many years under the title "Oxidation and Intermediary Metabolism", and for which no text has been available. Presupposing an elementary knowledge of the chemistry of carbohydrates, fats, and proteins and of the principles of physiology, it concentrates upon the transformations of organic food-stuffs within the body, and it is appropriate that it should be dedicated to Gowland Hopkins, who always insisted that this was indeed the central field of biochemistry. The book begins with an account of the nature and properties of enzymes, and a classified description of the major reactions catalyzed by them: this is distinguished by its orderly arrangement and the author's clear if not elegant style; the diagrams are ingenious and helpful The second half of the book deals with the intermediary metabolism of the amino-acids, carbohydrates, and fats, in turn. Here the author is at his best in describing such complex sequences of reactions as the glycolytic formation of lactic acid from glycogen, or the "citric acid" cycle, such as may be studied in a single organ or an extract; he is decidedly less successful in giving the reader a broad conception of the chemical activities of the body as a whole. There is no systematic account of the all-important isotope techniques and their results, and no emphasis on the picture they so vividly paint of the body as ceaselessly building up and breaking down its constituents, even those which are most rigid or complex and apparently permanent. At times, too, the author's interest in comparative biochemistry (on which he has written elsewhere) has tempted him to give space to rare compounds concerning whose "dynamic" metabolism little is known. Nevertheless the writing is continuously interesting, and the author has not hesitated to bridge gaps in our knowledge with some frank and plausible speculation; there are, however, certain major gaps of which he seems curiously unaware. The only bibliographic references in the book are to some fifty review articles, but there is an elaborate subject index. It should be made clear that the book is not written primarily for the medical man: disorders of metabolism are discussed only as they throw light upon the normal, the neuro-endocrine controls are hardly mentioned, and there is no account of the distribution and movements of water and salts; this is rather a description of intermediary metabolism and its background in enzymology.

Fundamentals of Clinical Neurology. H. H. Merritt, Professor of Clinical Neurology, College of Physicians and Surgeons, Columbia University; F. A. Mettler, Associate Professor of Anatomy, College of Physicians and Surgeons, Columbia University; and T. J. Putnam, Professor of Neurology and Neurological Surgery, College of Physicians and Surgeons, Columbia University. 289 pp., illust. $7.00. The Blakiston Co., Philadelphia and Toronto, 1947.

As stated in the preface, this book has been written to explain the essential facts which any practitioner of medicine must know concerning the problems of diseases of the nervous system commonly encountered in general practice. The book is divided into three parts dealing respectively with a practical technique for examination of the patient, the basic anatomical arrangements of the various parts of the neuro-axis and their functions, and finally by a description of the most commonly encountered disorders of the nervous system, including a summary of the findings in the cerebrospinal fluid in nervous diseases. The book will be of undoubted value to the graduate student but it is questionable if it will prove popular with the medical practitioner. The authors have given considerable space to describing the anatomy of various parts of the nervous system with the practical application in diagnosis. Attempting at the same time to be brief and concise, they have limited their description of disease entities, and undoubtedly this will impair the value of the book for the practitioner. For example, subacute combined degeneration is dealt with in less than one page while the structure and blood supply of the medulla receives seven pages. This emphasis on the anatomical and physiological aspects of neurological diagnosis will be very acceptable to

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the advanced student, but for the man engaged in practice its value will be limited.

The book has the great merit of clear, concise exposition, being an expression of the author's wide experience in neurology. There are a few mistakes which will doubtless be corrected in future editions. For example, in discussing confrontation testing of the visual fields, it is stated that the patient covers his left eye and looks at the examiner's right eye with his right eye while the right visual field is being tested. This is obviously incorrect and liable to prove confusing to the student. Errors of this kind are minimal, however, and the book can be highly recommended as readable and informative to all physicians who have a particular interest in neurology.

Modelling for Motherhood. D. H. Heinz and K. S. Bolt. 74 pp., illust. $2.00. John Wiley and Sons, Inc., New York; S. J. Reginald Saunders & Co. Ltd., Toronto, 1946.

This is a perfect example of presenting the practical and scientific facts of antenatal care, in non-medical terms. Filled with high good humour, this 74 page book carries the future mother through the months of her expectancy, and fully explains away the many new and strange sensations which occur during this period—much more efficiently, though lightly, than the physician seems able to do, with his unintelligible medical language.

Humorous, even occasionally grotesque illustrations accompany the text to stress the importance of controlled weight gain, and proper health care in pregnancy. Illustrated excerpts are included concerning clothing, and exercises, and a much needed list of baby's normal requirements for its first six months. The book is to be highly recommended to the doctor as a waiting room sedative and to the patient for home consumption.

Pathology of Labour, the Puerperium and the Newborn. C. O. McCormick, Clinical Profesor of Obstetrics, Indiana University School of Medicine. 514 pp., illust., 2nd ed. $9.50. The C. V. Mosby Co., St. Louis; McAinsh & Co., Ltd., Toronto, 1947.

This is an outgrowth of lectures prepared by the author for his senior medical students and it has the merits and demerits of such a method of presentation: the didactic approach, the clipped terse style, and the attempt to cover, in short compass, a very wide range. Probably for most readers and in dealing with a branch of medicine in which mistakes or failures of judgment are likely to prove tragic, such a method of presentation is desirable. In the three years since the advent of the first edition much that is new has appeared in obstetric literature and the digested material is to be found in the new volume. Topics such as the use of anti-coagulants in the management of thrombophlebitis, the problem of diabetes mellitus in pregnant women and the treatment of their offspring, early rising, the value of the sulfonamides, penicillin and streptomycin, the Rh factor and erythroblastosis fetalis have been faithfully discussed. Dr. McCormick shows himself a sound teacher and a safe guide.

Occupational Diseases of the Skin. L. Schwartz, Medical Director, United States Public Health Service; L. Tulipan, Clinical Professor of Dermatology and Syphilology, New York University, College of Medicine, New York City; and S. M. Peck, Dermatologist, Mt. Sinai Hospital, New York City. 964 pp., illust., 2nd ed. $12.50. Lea & Febiger, Philadelphia; The Macmillan Co. of Canada, Toronto, 1947.

The scope of this book is encyclopædic. Since the first edition appeared in 1939, when it rapidly assumed the leading position in works devoted to this subject, many new developments have appeared, which in the second edition receive full and detailed consideration. Many of these are direct consequences of the World War. New industries have been called into being, old ones have expanded beyond their originally conceived limits, new processes have developed in both old and new industries, new chemical compounds have been introduced into the industrial and also the domestic environment, and as a consequence of all this many new hazards exist. Workmen in numerous occupations are exposed to occupational skin injuries hitherto unknown. The current literature has covered these matters well, but the collection and arrangement of the new knowledge, with its application, has been excellently performed by Schwartz and his collaborators. Thirty new illustrations and a coloured frontispiece have been added with advantage. The latter is significant in that it illustrates chloracne, a subject discussed in connection with cutting oils, cable insulation and paper manufacture, which in the first volume received a mention of only one line. One additional mention may be made of the chapter on dermatitis from wearing material, which has been added, this being but one of the many new hazards to which modern skins are subjected.

This important book is an indispensable item in the library of dermatologists and all others interested in industrial medicine or compensation work. All engaged in general practice will find it of greatest value.

Principles and Practice of Obstetrics. J. B. DeLee, Consultant in Obstetrics, the Chicago Lying-in Hospital and Dispensary; and J. P. Greenhill, Attending Obstetrician and Gynæcologist, the Michael Reese Hospital. 1,011 pp., illust., 9th ed. $11.50. W. B. Saunders Co., Philadelphia and London; McAinsh & Co. Ltd., Toronto, 1947.

The changes which have been made in this edition need to be carefully evaluated, since for many years it has been a leading instrument in the teaching of obstetrics. A complete revision has been carried out, along the lines begun in the previous edition. Each chapter is followed by a complete bibliography of its contents so that advanced reading beyond the scope of the text is made readily available. The large number of excellent sketches and photographs which have been a feature in past editions has been increased still more. Each section has been brought up to date following the trend of modern thinking in obstetrics. Occasionally, opinions are expressed or quoted which are not as yet accepted as proved. They are few however, and detract very little from the book's value.

The cornerstone of the text continues to be that portion which deals with operative obstetrics. The classical descriptions of former editions have wisely been allowed to remain unchanged. Additions have been made with benefit to the portion dealing with Cæsarean section. Incidentally the strips from De Lee's motion picture of the forceps operation remain in this edition, as in at least the last, numbered improperly. The sequence of the pictures from these strips is vertical, while that of the numbers is horizontal.

One is impressed by the fact that this book has largely changed in character. The hand of De Lee has been almost completely replaced by that of Greenhill. This new almost completely rewritten edition is an exhaustive review of obstetrical literature woven around the dogmatic opinions of the author. The only remaining evidence of De Lee is found in the title, and in the retention of his unsurpassed descriptions of obstetrical management and operations. As a result, there emerges a textbook which is an improvement over the older editions as an ideal teaching instrument. It also becomes more of a consulting textbook, suitable for reference by specialists and general practitioners alike.

Recent Advances in Clinical Pathology. By various authors. Produced under the Auspices of the European Association of Clinical Pathologists. General Editor S. C. Dyke, Pathologist, the Royal Hospital, Wolverhampton. 468 pp., illust. $6.25. The Blakiston Co., Philadelphia and Toronto, 1947.

This volume is a further addition to the number of medical publications listed in the Recent Advances Series. As it is a first edition the advances which



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may be accepted as recent had to be selected arbitrarily. Another factor in making portions of the book appear somewhat archaic was the delay between the inception of the work and its appearance in print. For this delay and for any defects in the book the editor generously accepts full responsibility. Even in times of peace much stamina is needed for the effort of editing a publication which has forty contributors and the trials of the post-war period made such an effort almost heroic. The final verdict, however, must be that he has done his task well and should rightfully be given credit for the coherence, consistency and compactness of the work. The writing is naturally varied but it is generally clear, readable and with little redundancy and no contributor has drifted far into prolixity. The opinions expressed are rarely unorthodox and the appraisals of the helpfulness and applicability of most tests now in clinical use are temperately judicious. Although the book's foremost place will be as a source reference for those working in hospital laboratories, practitioners, both specialist and general, will find it a dependable guide when seeking diagnostic aid from the laboratory procedures available in a well-equipped hospital.

The popularity of the work may be less than is due to its intrinsic merit because under the term Clinical Pathology much is included which, on this side of the Atlantic, is looked on as Biochemistry. A competent biochemist has such an important place in modern medicine that, perhaps not unreasonably, he will be indisposed to welcome with enthusiasm anything which suggests that he is a mere appendage of some other department in the hospital or in the medical school.

Rotunda Hospital, 1745-1945. O. T. D. Browne, Ex-assistant Master and L.M. Rotunda Hospital, Dublin. 296 pp., illust. $9.50. E. & S. Livingstone Ltd., Edinburgh; The Macmillan Co. of Canada, Toronto, 1947.

The publication of this book coincides with the recent celebrations of the bicentenary of the famous Rotunda Hospital. As an ex-assistant Master of the Rotunda and a student of historical medicine Dr. Browne is thoroughly qualified to write the story. It is a well documented work with no foot notes but a list of references at the end of each of the main sections, which makes for easy reading.

The primary object of Bartholemew Mosse, the founder, was to bring relief to the poor lying-in women of Ireland. Throughout the two centuries of its existence the Rotunda has been faithful in this respect. In addition it has become a great teaching centre of obstetrics and gynæcology, attracting students and physicians from all over the world. In consequence the book deals not only with the history of the institution, but also with the progress of obstetrics and gynæcology. Sometimes the Rotunda lagged behind, sometimes it was in the van as in Tweedy's treatment of eclampsia, but its influence was never negligible. The fact that the tenure of office of each master is limited to seven years is probably a factor in making the institution dynamic rather than static.

In a work characterized by careful scholarship it is unfortunate that there are a few minor lapses. The apochryphal story of Sir Walter Scott suggesting a coat of arms for Sir James Y. Simpson is repeated, though Scott died in 1832 some thirty-four years before Simpson received his baronetcy. On page 175 the statement is made "The condition of the incarceration of the retroverted gravid uterus was first recognized, and successfully treated by Marion Sims of South Carolina (1813-1888)". Though Sims may have been the first to treat successfully incarceration of the retroverted gravid uterus, the condition is beautifully illustrated in William Hunter's "Anatomy of the Human Gravid Uterus" published in 1774. Plate 26 in that work has the following inscription: "from the tenth subject on the fifth month showing the circumstances of a retroverted womb". In the chapter on puerperal fever proper credit is given to Holmes (1843) and Semmelweiss (1861), but no mention is made of Alexander Gordon of Aberdeen who in 1795 proclaimed the doctrine of the contagiousness of puerperal fever.

In the difficult task of assessing the Masters, Dr. Browne picks out as outstanding—Mosse, Ould, Clarke, Labatt, Collins, Macan, Smyly and Tweedy. Mosse he considers to be the greatest since he had the courage to dream of giving assistance to poor women in labour and the initiative to make his dream come true.

Sensory Mechanism of the Retina. R. Granit, Director of the Nobel Institute for Neurophysiology. 412 pp., illust. $10.00. Oxford University Press, London; McAinsh & Co. Ltd., Toronto, 1947.

The author of this most interesting and informative book is a world authority on electrophysiology, with particular reference to the application of electroretinography to the solving of many problems in visual perception. For many years he and his co-workers at the Royal Caroline Institute, Stockholm, have carried out fundamental research in this field. The present volume represents the summing up of the work to date, and its correlation with the findings of others in this and related studies. This volume will hold its greatest interest for the specialist in electrophysiology and for the academic physiologist. For students in the field, it may well become the reference work of choice. By others with not so specialized interests it will be read with interest and improvement. Professor Granit has a most lucid and readable style. The subject matter is well organized, and the illustrations well chosen and reproduced.

Signs and Symptoms. Edited by Cyril Mitchell MacBryde, Assistant Professor of Clinical Medicine, Washington University School of Medicine. 439 pp., illust. $14.00. J. B. Lippincott Co., Montreal and Philadelphia, 1947.

This book, from the pens of Dr. MacBride and twenty other contributors, is an attempt to assist the physician in the analysis and interpretation of the patients' complaints. The preface points out that great advances have been made in the various techniques of laboratory diagnosis but similar advances have not been made in the understanding of the meaning of symptoms. Emphasis is placed upon the pathologic physiology of the symptom rather than upon the correlation of the symptom with physical and laboratory evidence.

The volume is a practical approach to the physiopathology of disease likely to appeal to the practising physician interested in a better understanding of the disease processes with which he comes in daily contact. The book should also appeal to the undergraduate teacher.

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